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EUGENIC ASPECT OF CLEFT PALATE AND OTHER FACIAL DEFORMITIES

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AT this time, when every available ounce of human endeavor is essential for our war effort, the many lives wasted because of facial and oral deformities should and can be restored to usefulness. An understanding by the medical profession and the lay public is essential for further development of the remarkable results that surgical and dental prosthesis has accomplished.

These wasted lives present a twofold problem: (1) the obvious factor of unusable potential man power; (2) the more insidious and serious factor of morale. During the first World War, the number of facial and oral mutilations was so great that even at this late day there is existent a French society of mutilated war veterans. The appearance of these veterans on the streets of Marseilles and Paris had such a demoralizing effect on civilian and military morale that it was believed to be a direct contributing factor in the second revolt of the French Army in 1918.

In a report from the War Department on "Defects Found in Drafted Men," in 2,500,000 men examined there were 1,183 cases of cleft palate and 283 cases of cleft lip. Vermont showed the highest percentage of cleft palate, 1.55 per thousand. These figures relate only to men between 21 and 32 years of age examined. When we consider the fact that women of the same age would probably show the same percentage; that, in the younger stages of life, the percentage is much higher and, furthermore, the large number of both sexes under 21 and over 32 years with palatal defects, we cannot but realize that the percentage of cleft palate patients is higher than was shown by the War Department.

Now we are again at war. With the certainty of many serious oral and facial wounds among the war casualties, requiring skilful maxillofacial prosthesis, this important work will undoubtedly write a new chapter in the history of American Dentistry. However, this spe-

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cialty is still ignored for the most part, in the undergraduate as well as the postgraduate dental schools.

At the 1942 meeting of the International Association for Dental Research, an exhibit was held at the School of Dental and Oral Surgery, Columbia University. Case after case was exhibited, showing ruined and deformed mouths, distorted and mutilated faces that had been restored to normal by the use of artificial appliances. Besides photographs, actual appliances restoring noses and ears, and also palatal obturators and mandibular appliances were exhibited.

Those of us who are familiar with this work recognize the fact that the individual handicapped by oral or facial deformities, whether congenital or acquired through civilian or war injury or disease, can be restored to normal status in society. Often when there has been extensive destruction and oral and plastic operations cannot restore the tissues completely, the last resort is surgical dental prosthesis. This branch of dentistry aims to reconstruct congenital and acquired defects of the mouth and face by appliances intimately associated with surgical treatment and living sculpture. Thus, the often forgotten unfortunates may again become useful members of society and make their contribution to the common welfare.

In the realm of congenital facial defects, cleft lip and cleft palate are the most common. Each year, thousands of patients eagerly awaiting the arrival of a child are thrown into despair when they first see their infant. There may be one or two deep gashes running the full length of the lip to the distorted nose. When the infant attempts to suck, the liquid escapes through the nose. The attending physician informs the parents that the child was born with a cleft lip or cleft palate, or both. The mental and moral effect of this affliction is one of social maladjustment, which usually results in a sense of inferiority and frustration and not seldom in delinquency and criminal or psychopathic tendencies. A recent article[17] reports a case that reads like fiction, a tragic case in which society stepped in too late, with too little. Now society must pay too much.

In the cleft palate patients, the clefts may vary in size from a small opening in the hard palate to a complete cleft of the entire roof of the mouth, invariably accompanied by a single or double cleft in the lip. One child in 10,000 to 12,000 is born with this deformity. In New York State, there are approximately 11,000 people who were born with this defect.

A study of the birth records of 202,501 children born in Pennsylvania in 1942 revealed that one in 800 had a cleft palate. Of these 250 infants having cleft palate, 155 were boys, eighty-eight girls.

These malformations are brought about by defective fusion or complete absence of fusion between certain structures during embryonic life. The specific cause is still unknown. The following are possible causes for this condition:

1. Improper nourishment of the embryo. Recent experiments point toward lack of vitamin A.
2. Heredity.
3. Mechanical influences. Pressure from embryo knee, tongue or hand may tend to hold the uniting processes apart.
4. Poisonous substances circulating in the expectant mother: drugs, alcohol and toxins.

The competent surgeon repairs the lip several weeks after birth and usually operates on the palate when the child is about 3 years old. The results in most cases are remarkable. However, there are many cases wherein the health of the child is poor and the palatal operation may never be performed. Sometimes, even after a skilful operation, the palate is only partially closed or there may be a shortening of the uvula because of the initial extent of the abnormality. These children have difficulty with their speech

and their food passes through the nose as they attempt to swallow. Thus patterns of inferiority soon develop, often preventing them from taking their rightful position in society.

The rôle of physical attractiveness in sexual charm is recognized by students of eugenics. Knight Dunlap, among others, believes that beauty is the predominating element in mate selection. Whether this is so or not, the fact remains that the child, male or female, and particularly the latter, born with cleft palate starts life with a severe social handicap. Correction of the deformity and prevention of the psychic inferiority that comes with the defect in speech and appearance convert a eugenic liability into a eugenic asset. The interest of eugenics lies in the fact that a young woman or man who has had a cleft lip or cleft palate properly corrected is much more likely to mate with a superior person and produce superior children than the unfortunate individual in whom the defect has been permitted to persist, either through negligence born of ignorance or through inefficient surgical treatment and after-care.

The question of heredity must be considered. There is no reason for fearing that the children of a person having a cleft palate will necessarily be born with the same defect, though it is natural that the chances are greater than with normal parents. The latest statistics on the subject indicate that approximately 80 per cent of children born with this defect come from normal parents without any history of the defect in the family line. In the remaining 20 per cent, there is such a history.

The probability that the child with cleft lip or cleft palate will become a normal person is excellent, provided the parents will do their part faithfully and intelligently. This requires a program which involves strict supervision for several years, first by the surgeon, then by the dentist, the nurse, the speech expert, the family doctor and the family itself. This program must be begun on the day that the child is born. The first problem is that of feeding and stimulation of the natural reflexes of sucking and swallowing. Feeding will be easier after the lip has been surgically repaired.

Unfortunately, the facilities for such a complete program of rehabilitation are not within the means of families of moderate or low income. There is an urgent need for an institution or clinic in which the infants of these low income groups can be cared for with the same facilities as are available to people of greater means.

The rôle of the operating surgeon is a responsible one. Only a surgeon with experience in cleft palate surgery should be entrusted with the task of restoration. Merely to close the gap, as is often done, is not enough. The surgeon must be a facial sculptor. He must as nearly as possible insure a normal and attractive facial appearance. Both utility and beauty should be the constant aim. The closure must be made in such a manner as to prevent tissue strain and stress, which may result in an increase of the abnormality as the child grows older. He also must create a functioning soft palate.

If the oral surgeon realizes that the operation has not completely closed the opening in the palate, he refers the patient to the specialist in dental prosthesis, who is able to restore the missing tissues by obturators and other dental appliances. These ingenious devices not only close the opening, but they also simulate the missing tissues in their proper physiologic and biologic function. Then, with the proper speech training, the patient makes rapid strides toward a normal life.

In addition to cleft palate, the prosthetic dentist is also called upon to restore parts of the face and the oral cavity that may have been destroyed by disease: tuberculosis, tumors or cancer, x-ray burns, industrial accidents, gunshot wounds and

war injuries. The nose and ears and other parts of the face are replaced by special latex devices, sculptured to resemble the original parts and colored to blend with the skin of the individual. Thus we see the prosthetic dentist as the right arm of the oral surgeon in these seemingly hopeless cases.

It is unfortunate that this specialty has not attained its rightful place in the medical and dental fields, because no leader has arisen from the ranks to organize this field as an important specialty in dentistry, and because the patients are generally of insufficient means to make this difficult work lucrative for the average practitioner. Truly, one must be a humanitarian to be attracted to this important field. At the present time, there are not sufficient dentists trained to care for the civilian population with these defects. How can the war casualties be cared for?

As stated before, there is an urgent need for a clinic where these facial deformities can be properly handled, not only where the operation can be performed by surgeons specially skilled and trained in this work, but also where the parents can be instructed and guided in the various stages of the child's growth and development up to adolescence and maturity. One can visualize such an institution, which will keep the child under constant observation and sympathetic care from the day of birth until the child matures and takes his rightful place as a useful member of the community. From the eugenic standpoint, society has much to gain by this rehabilitation of the facially deformed and their restoration to the normal. Although it means much effort and patience, it can be done. It has been done and there is every reason that it should be done.

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THE SYSTEMIC INFLUENCE UPON BONE IN PERIODONTOCLASIA*

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INTRODUCTION

MOST of the studies in periodontoclasia have sought an explanation for the bone loss which occurs in this condition. This emphasis on bone loss is a logical one because, in the final analysis, it is the destruction of supporting bone which is responsible for the mobility of the teeth and their ultimate loss in periodontoclasia. The literature presents three major concepts regarding the basic nature of this bone loss. The first concept (local) is that periodontoclasia is purely a local inflammatory process, produced by irritants in the oral cavity, in which the bone loss results from the inflammation. According to the second concept (systemic), the bone loss in periodontoclasia originates as a non-inflammatory atrophic or degenerative change which is the result of purely systemic influences. The third concept (compromise) maintains that there are three different types of periodontoclasia, the local and systemic types mentioned above and an additional type which represents a combination of the two others. Since extensive quotation from all the papers touching on this subject would do nothing to clarify this presentation, only those investigations which seem to us to be the most pertinent will be referred to here.

The local concept[1, 2] is most clearly stated by James and Counsell,[3] who, after studying periodontoclasia in human necropsy material, believe "the disease to be local in origin" and that "it commences as a gingivitis," which is "followed by progressive marginal absorption of bone."

The systemic concept is presented by Hopewell-Smith,[4] who maintains that "pyorrhea alveolaris does not begin as a gingivitis," but "is essentially dependent upon an osseous lesion, an atrophy of the bone." Also, according to Fleischmann and Gottlieb,[5] "atrophy of bone is the primary factor in the etiology in pyorrhea," and "the atrophy of bone is surely due to a constitutional factor." In Roy's[6] opinion of periodontoclasia, "precocious senile atrophy is its primary lesion."

The compromise concept,[7, 8, 9] as stated by Gottlieb and Orban,[10] is that "two factors, entirely independent of each other, may therefore cause the formation of a deep crevice," one being entirely local *(Schmutzpyorrhea)* and the other constitutional (diffuse atrophy). Gottlieb[11, 12] and Orban and Weinmann[13] have presented the histologic details of the condition recognized as the systemic type of periodontoclasia. In the opinion of Thoma and Goldman,[14] there is a local type of periodontal disease (parodontitis) "in which the first changes occur in the gingivae and the bone becomes involved later," a systemic type

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*Prize winning essay in the Third Annual Prize Essay Competition sponsored by the Chicago Dental Society; read before the first general session of the Eighteenth Annual Midwinter Meeting of the Chicago Dental Society, February 21, 1944.

(parodontosis) in which "the initial lesion . . . is in the alveolar bone around the teeth" and a combined type (syndrome) which results from a combination of parodontitis and parodontosis.

The foregoing opinions reveal a general acceptance of the concept that bone loss in periodontoclasia is associated with gingival inflammation. It should be noted, however, that any conclusions drawn from necropsy study, such as those quoted above, are subject to the limitations of necropsy studies in general; that is, they present a static picture of tissues at the time of death, but do not give full indication of causal relationships. The latter information can come only from correlating histologic findings with the experimental or clinical conditions which produced them. Thus, in the periodontal field, study of necropsy material cannot establish whether the bone resorption associated with gingival inflammation is a result of that inflammation or the cause of the inflammation, or the extent to which systemic influences are responsible for either or both of these findings.

Experiments, it is true,[15, 16, 17, 18, 19, 20, 21] have demonstrated that bone loss occurred in the jaws when a generalized skeletal disturbance was created in animals. However, since the systemic background was the only condition which was artificially altered in these experiments, it was not possible to determine the degree of interdependence of local and systemic influences in periodontal bone loss.

It was believed that if a series of animal experiments were arranged so that it would supplement previous studies and fill in gaps in our knowledge which have served to divide the opinions of investigators in the past, the controversy which exists regarding the basic nature of bone loss in periodontoclasia might be settled. The object of such experiments would be to determine whether altering the systemic background would affect the nature of changes seen in the bone beneath inflammation areas in the gingival crevice and what the nature of this effect upon the bone would be. To accomplish this, it would be necessary, first, to induce inflammation in the gingival crevice in a group of normal animals and in a group of animals in which there was a generalized skeletal disturbance of systemic origin; second, to determine whether and in what manner the jaws were affected by this skeletal disturbance; third, to establish the histologic details of the bone loss beneath the area of artificially induced inflammation in the normal group, and fourth, to determine how the details of the bone

Fig. 1.—Amalgam in interproximal space between first and second mandibular molars.

loss beneath the area of artificially induced inflammation in the normal group were altered by the local effect of the generalized skeletal disturbance.

EXPERIMENTAL PROCEDURE

Thirty adult male albino rats, Wistar strain, from 3 to 4 months old, the average weight of which was 250 gm. were used as experimental animals. In order to induce inflammation in the gingival crevice, freshly prepared amalgam was packed into the interproximal space between the first and the second mandibular molars in the anesthetized animals. The amalgam was allowed to remain in the interproximal space for experimental periods of from seven to thirty days. (Fig. 1.)

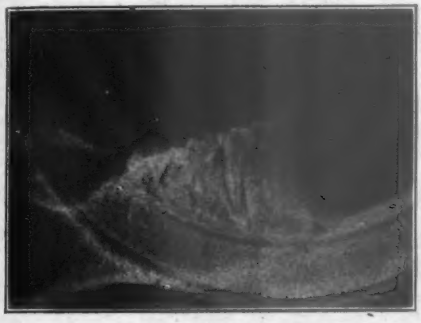

Fig. 2.—Normal diet group; untreated mandible. The radiolucent space between the first and second molars is the result of normal contours of proximal surfaces.

Fig. 3.—Normal diet group, animal 6; showing reduction of height of interdental septum below amalgam in interproximal space. (The radiopaque object superimposed on the distal root of the first molar is a piece of amalgam in the mucobuccal fold.)

Immediately after the above-described treatment, the animals were divided into two groups. One group of fifteen animals was fed a stock diet of Purina Dog Chow Checkers, supplemented by fresh lettuce and cabbage (normal diet group), and the remaining fifteen animals were deprived of all food, but were allowed water *ad libitum* (starvation group).

At the end of from seven to thirty days, all the animals were killed and the jaws, tibiae, ribs and vertebrae were removed and roentgenographed. These bones were then fixed in formalin, decalcified in formic acid solution and embedded in paraffin.

Fig. 4.—Starvation group, animal 23; showing reduction of height of interdental septum below amalgam in interproximal space.

ROENTGENOGRAPHIC FINDINGS

X-ray study revealed reduction in the height of the interdental septum beneath the amalgam in both the normal diet group (Figs. 2 and 3) and the starvation group (Fig. 4).

MICROSCOPIC FINDINGS

The bone findings are arranged in three sections:

Section 1. A comparison of the tibiae, ribs, vertebrae and mandible of the starvation group with one another and with the same bones in the normal diet group.

Section 2. A comparison of the interdental septa subjacent to gingival crevices uninvolved by artificially induced inflammation in the normal diet group with the same regions in the starvation group.

Section 3. A comparison of the interdental septum subjacent to the artificially induced inflammation in the normal diet group with the same area in the starvation group.

By arranging the microscopic findings in this manner, it will be possible to establish the following:

(a) Whether there is any similarity between bone changes in the mandible and those in bones elsewhere in the skeletal system as the result of a systemic disturbance.

(b) How the periodontal bone is af-

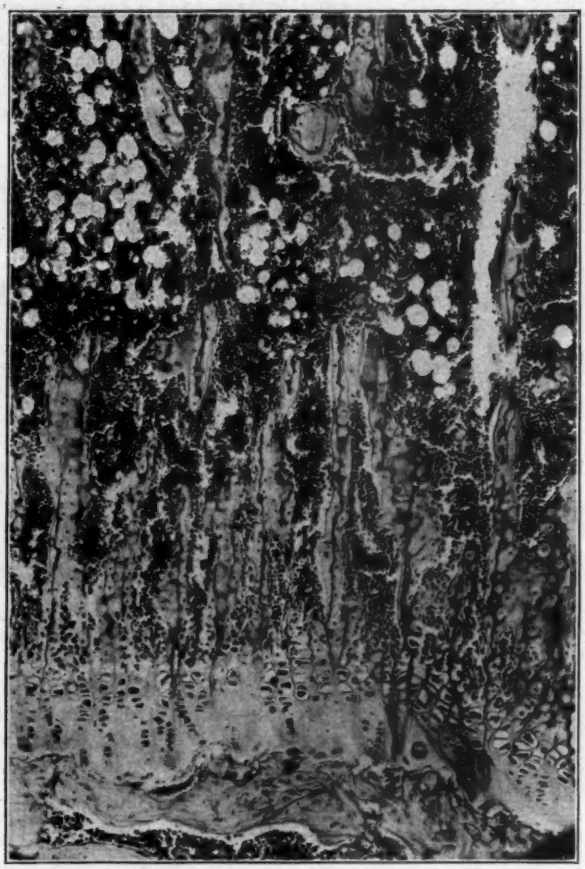

Fig. 5.—Normal diet group, animal 7; head of tibia below epiphyseal cartilage showing bone trabeculae containing remnants of cartilage and bordered by pale zone of new bone matrix. (Hematoxylin and eosin; ×96.)

fected by a generalized skeletal disturbance.

(c) What effect gingival inflammation has upon the underlying bone in the absence of a generalized skeletal disturbance.

(d) How the introduction of a systemic disturbance would alter the effect of gingival inflammation on the underlying bone.

Section 1. *A comparison of the tibiae, ribs, vertebrae and mandible of the starvation group with one another and with the same bones in the normal diet group*

Tibiae, ribs and vertebrae

Normal diet group*

In the head of the tibia immediately below the epiphyseal cartilage and in the rib at the osteochondral junction, the bone was arranged in parallel continuous trabeculae included within which were remnants of cartilage (Fig. 5). The borders of the trabeculae were formed by pale pink bone matrix along which beaded strands of osteoblasts were observed in some areas.

In the body of the vertebrae adjacent to the spinal cord, the bone trabeculae were lamellated and presented regular smooth margins (Fig. 7).

*Throughout the skeletal system, the animals in this group presented what is believed to be a normal picture.

Starvation group

The bone below the epiphyseal line in the head of the tibia and at the osteochondral junction in the rib was arranged in discontinuous thin trabeculae. There were no osteoblasts or deposits of pale pink matrix along the trabecular margins. Instead, the borders of the trabeculae were for the most part irregularly roughened and fragmented (Fig. 6).

In the body of the vertebrae adjacent to the spinal cord, the bone trabeculae were thin so that the marrow spaces were enlarged in comparison with the normal. In some areas, the continuity of the trabeculae was broken and the bone terminated in abrupt roughened ends. In addition, there were areas in which the trabecular margins were eroded and roughened (Fig. 8).

Mandible between the bifurcation of the molar roots

Immediately below the cementum in the bifurcation of the molar roots, a pale pink zone of bone lined with osteoblasts was observed. This bone matrix lacked the lamellations seen in the underlying trabeculae to which it was attached. The borders of the underlying trabeculae were formed for the most part by a linear layer of pale pink matrix (Fig. 9). In a few scattered areas, irregular concavities were seen along the trabecular margins.

There was no evidence of pale pink bone matrix or osteoblasts along the bone trabeculae in the bifurcation of the roots. The trabecular borders were irregularly eroded and comprised of continuous irregular hollowed-out concavities. In addition, the bone trabeculae were thinned and presented areas in which the lacunae were empty and enlarged. The matrix was poorly stained and fragmented at the roughened periphery (Figs. 10 and 11). These changes resulted in a loss of bone structure and an increase in the width of the periodontal membrane.

Mandible: vascular channels and marrow spaces

Normal diet group

Vascular channels extended from the periodontal membrane into the adjacent bone and were also seen in the remainder of the jaw. The lining of the vascular channels and marrow spaces were similar in appearance. They were lined in part with a dis-

Starvation group

The vascular channels and marrow spaces throughout the jaw were increased in width and size. The lining of these regions was formed for the most part by irregular continuous concavities interspersed among which were relatively few small areas with

tinct border of pale pink matrix along which a single layer of polyhedral osteoblasts was observed (Figs. 9, 12 and 14). The remainder of the lining was devoid of pale pink matrix and in addition presented sections formed by hollowed-out concavities containing ovoid connective tissue cells.

smooth linear basophilic margins. The concavities presented a roughly eroded granular border formed by discrete minute eosinophilic particles which blended with the remainder of the bone matrix (Figs. 10, 13 and 15). Multinuclear osteoclasts were a common finding in these concavities. The bone lacunae at the periphery of the vascular channels and marrow spaces were enlarged and devoid of osteocytes. In some regions, open enlarged irregular osteocytic lacunae formed part of the margin of the channels and marrow spaces.

Section 2. *A comparison of the interdental septa subjacent to gingival crevices uninvolved by artificially induced inflammation in the normal diet group with the same regions in the starvation group*

Interdental septa in the absence of inflammation

Normal diet group

The mesial surface was formed for the most part by small contiguous concavities, the majority of which contained connective tissue cells with spherical nuclei and connective tissue fibers. In a few of the concavities, multinuclear osteoclasts were observed in close apposition with the minutely roughened bone margin. Other concavities contained connective tissue cells with prominent ovoid nuclei in apposition with a pale pink matrix of varying thickness. This matrix was demarcated from the remainder of the bone by a deeply basophilic line which outlined the irregular concavity margin (Fig. 16).

The distal surface presented a moderately smooth margin formed for the most part by a border of pale pink matrix which was lined with polyhedral connective tissue cells with large spherical nuclei (Fig. 16). Oval lacunae containing a large prominent basophilic spherical nucleus were observed in the pale pink martix. The bone matrix along the surface was demarcated from the remainder of the bone by a regular fuzzy basophilic line which was parallel to the long axis of the adjacent root. At the periphery, several cells were observed which were partially embedded in matrix and partially surrounded by the adjacent connective tissue.

The gingival crest presented a smooth rounded border which enclosed a pale pink homogeneous matrix containing lacunae with deeply staining spherical nuclei (Fig.

Starvation group

The mesial surface was formed in its entirety by large irregular concavities with roughly eroded margins. In addition, the bone matrix at the periphery was fragmented. The marginal concavities were of greater diameter and extended for a greater depth into the septum than was the case in the normal group. Multinuclear osteoclasts observed in the normal group were present, but not increased in number along the mesial surface (Fig. 17).

The distal surface was formed for the most part by a fuzzy pink bone matrix which was fragmented at the periphery and blended with the adjacent connective tissue (Fig. 17). There were only occasional small scattered zones in which the margin was formed by a clearly staining eosinophilic matrix with a smooth border and a lining of polyhedral osteoblasts similar in appearance to that observed in the normal. The osteocyte lacunae at the periphery were surrounded in part by the bone matrix and in part by filamentous eosinophilic connective tissue fibrillae.

Loss of bone structure was observed at the gingival crest in the starvation group. There was fragmentation of the bone matrix at the periphery with the formation of an irregular sawtooth edge (Figs. 18, 19 and 20). The matrix was markedly granular in appearance and blended with the surrounding connective tissues. The osteocyte lacunae were enlarged and, in many instances, were devoid of osteocytes. Enlarged open

16). Prominent connective tissue fibers which extended from the bone were interspersed between ovoid connective tissue cells at the margin. Two or three small contiguous concavities containing connective tissue cells was an occasional finding at the distal portion of the gingival margin.

lacunae from which the osteocytes were missing formed irregular concavities at the bone margin.

Section 3. *A comparison of the interdental septum subjacent to the artificially induced inflammation in the normal diet group with the same area in the starvation group*

Gingival crest of interdental septum subjacent to experimentally produced inflammation in the gingival crevice

Normal diet group

There was loss of bone structure and a dense infiltration of lymphocytes in the connective tissue bordering the bone margin. The bone margin presented many irregular continuous concavities with minutely roughened borders (Figs. 21 and 22). The concavities contained ovoid connective tissue cells. Adjacent to these concavities and immediately subjacent to the inflammatory lymphocytes and plasma cells, the bone margin consisted of a smooth pale eosinophilic layer of bone along the border of which was a single row of polyhedral connective tissue cells. There was no alteration in the bone matrix or bone lacunae subjacent to the margin.

Starvation group

There was loss of bone structure and a dense infiltration of lymphocytes in the connective tissue bordering the bone margin. The bone margin was comprised of irregular continuous concavities of varying size, a few of which contained multinuclear giant cells (Figs. 23 and 24). The bone matrix at the margin was markedly granular and presented peripheral fragmentation. For a considerable depth below the margin, the osteocyte lacunae were enlarged, with irregular contours, and were devoid of osteocytes. Serial sections revealed large sections of bone which were segregated from the remainder of the interdental septum and separated from it by multinuclear giant cells (Fig. 25).

INTERPRETATION OF FINDINGS

This series of animal experiments has established basic principles which govern the bone changes in periodontoclasia. While it is true that these principles have been suggested in part by previous investigation, the controlled conditions supplied in these animal experiments have, for the first time, made it possible to separate and weigh independently the influence of local and systemic factors in periodontoclasia. By correlating our conclusions with the findings of clinical experience, it becomes possible to clarify some of the controversial points relating to the destruction of periodontal bone and to suggest a more rational approach to periodontal treatment. This is attempted in the following sections (I-IV).

I. The bone of the jaws undergoes changes similar to those which occur in other bones as the result of generalized skeletal disturbances of systemic origin. This is revealed by the finding of osteoporotic changes in the tibia, rib and vertebrae of each animal in the starvation group in addition to similar changes in the mandible. Bone formation which was observed in the mandible of the animals in the normal diet group was, with minor exceptions, absent in the starvation group. In the latter group, the bone trabeculae in the tibia, rib and vertebrae were also thinned and often fragmented, and although there was some bone deposition, it was markedly

reduced as compared with the normal group. The composite picture in the starvation group was one of generalized skeletal osteoporosis with marked predominance of bone resorption and a negligible degree of normally existent bone formation.

Fig. 6.—Starvation group, animal 23; head of tibia below epiphyseal cartilage showing bone trabeculae devoid of bordering zone of pale new matrix and presenting roughened and fragmented margins. (Hematoxylin and eosin; ×96.)

The sensitivity of the bone of the jaws to generalized skeletal tendencies revealed above is clinically significant. It emphasizes the futility of evaluating periodontoclasia in terms of local influences alone. Since the bone of the jaws reflects the prevailing condition of the skeletal system, changes in the periodontal bone are constantly subject to a systemic regulating influence. This systemic regulatory influence, which is active in health and disease, constitutes a "bone factor" which is fundamental in all cases of periodontoclasia.

II. In the absence of chronic gingival inflammation, bone loss may occur in periodontoclasia as a result of general-

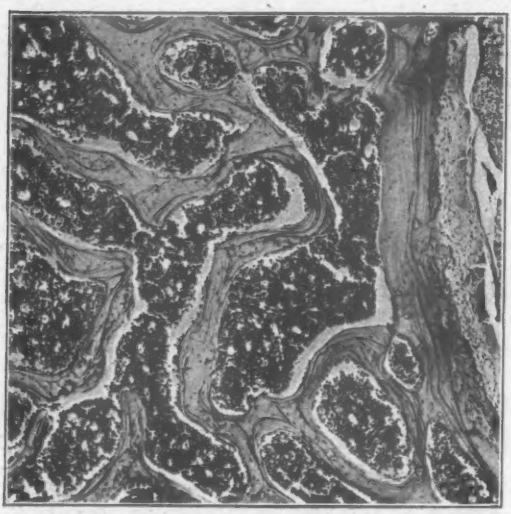

Fig. 7.—Normal diet group, animal 6; body of vertebra showing lamellated bone trabeculae with regular smooth margins. (Hematoxylin and eosin; ×89.)

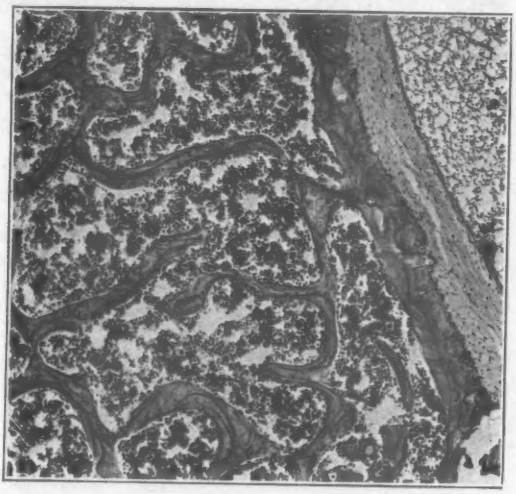

Fig. 8.—Starvation group, animal 23; body of vertebra showing thinned bone trabeculae and enlarged marrow spaces. Areas in which the trabecular margins are eroded and roughened are also seen. (Hematoxylin and eosin; ×89.)

ized skeletal disturbances. A reduction in the height of the interdental septa subjacent to gingival crevices unaltered by inflammation was observed in the starvation group. The height of the interdental septa represents the result of an equilibrium between bone formation and bone resorption. In the normal diet group, this equilibrium is regulated so that the height of the septa is main-

Fig. 9.—Normal diet group, animal 6; bone in bifurcation of molar roots showing pale pink zone of bone lined with osteoblasts beneath cementum. The underlying trabeculae are for the most part lined with pale bone matrix. A portion of the trabecular border is formed by continuous irregular concavities. (Hematoxylin and eosin; ×146.)

Fig. 10.—Starvation group, animal 9; bone in bifurcation of molar roots showing peripheral resorption and fragmentation of bone matrix. There is loss in bone substance and thickening of the periodontal membrane, with absence of bone formation. (Hematoxylin and eosin; ×140.)

tained. In the starvation group, the balance of the equilibrium is disturbed because bone formation is markedly reduced and bone resorption predominates. Bone substance is lost as the result of this altered bone equilibrium in the starvation group and the height of the interdental septa is reduced. In addition

Fig. 11.—Starvation group, animal 10; bone in bifurcation of molar roots showing resorption lacunae along trabecular borders. There is fragmentation of the bone matrix immediately below the cementum, with absence of bone formation along vascular channels. (Hematoxylin and eosin; ×120.)

Fig. 12.—Normal diet group, animal 7; marrow space. The bone bordering the marrow space is lined in part by a layer of pale pink matrix and in part by continuous shallow concavities. (Hematoxylin and eosin; ×68.)

to the altered bone equilibrium in the starvation group, fragmentation and dissolution of the bone matrix, with release of osteocytes, and the formation of connective tissue at the expense of the bone substance were also noted. These are responsible for the reduction in the height and diameter of the interdental septa.

Because reduction in the height of the interdental septa is a cardinal clinical and roentgenographic symptom of periodontoclasia, the above-described demonstration of its occurrence in the absence of local inflammation is significant. It accounts for the many cases of periodontoclasia which present roentgenographically marked bone loss in the absence of clinically detectible gingival inflammation.

III. Inflammation in the gingival crevice may result in bone resorption and reduction in the height of the interdental septa in the absence of generalized skeletal disturbance. Numerous necropsy studies and clinical observations have led to the generalized acceptance of this principle in the past. However, many details pertaining to it have been clarified in the experiments reported in this paper. Reduction in the height of the interdental septa was observed in the normal diet group beneath the artificially induced inflammation. This was indicated by the typical morphologic picture of bone resorption. The bone margin beneath the area of inflammation was formed in part by irregular continuous concavities of resorption. The remainder of the bone margin consisted of a smooth pale eosinophilic layer of bone bordered by a row of polyhedral osteoblasts. The underlying bone in the remainder of the septum was morphologically unaltered.

Fig. 13.—Starvation group, animal 9; marrow space. The bone bordering the marrow space is lined for the most part by extensive hollowed-out areas. There is an absence of bone formation. (Hematoxylin and eosin; ×68.)

The fact that such bone loss occurs in the absence of a generalized skeletal disturbance does not mean that it occurs independently of the influence of the prevailing generalized skeletal tendencies which constitute the bone factor in the individual animals. On the contrary, the degree of resorption which resulted from artificially induced inflammation in the normal diet group was limited by the generalized skeletal tendency toward bone loss subjacent to gingival inflammation is regulated by tendencies toward bone formation which are a phase of the normal skeletal pattern or "bone factor."

By revealing the microscopic details of bone loss subjacent to chronic inflammation in the absence of a generalized systemic disturbance, this phase of the experiments established a basis of measuring, by comparison, the local effect

Fig. 14.—Detail of Figure 12, normal diet group, animal 7; segment of bone in marrow space showing pale new bone matrix along border. This is separated from the remainder of the bone by an irregular line marking previous resorption. (Hematoxylin and eosin; ×360.)

bone formation in these animals. This was demonstrated by the zone of newly formed bone adjacent to the concavities of resorption along the bone margin subjacent to the inflammation.

It must, therefore, be recognized that, even in normal animals, the progress of bone loss subjacent to gingival inflammation is regulated by tendencies toward bone formation which are a phase of the normal skeletal pattern or "bone factor."

By revealing the microscopic details of bone loss subjacent to chronic inflammation in the absence of a generalized systemic disturbance, this phase of the experiments established a basis of measuring, by comparison, the local effect of introducing a systemic disturbance.

IV. The severity of bone loss subjacent to gingival inflammation is increased by generalized skeletal disturbances of systemic origin. It has been demonstrated above that bone loss occurred beneath areas of experimentally

produced inflammation in both the normal diet group and the starvation group. However, the details of the bone changes responsible for this loss were different in the two groups. The differences resulted from altering the systemic background. They are tangible indices of the extent to which the response of periodontal bone to gingival inflammation is affected by systemic conditions. The regulatory rôle of systemic influences upon the periodontal bone is emphasized by a comparison of the bone changes responsible for the bone loss in each group.

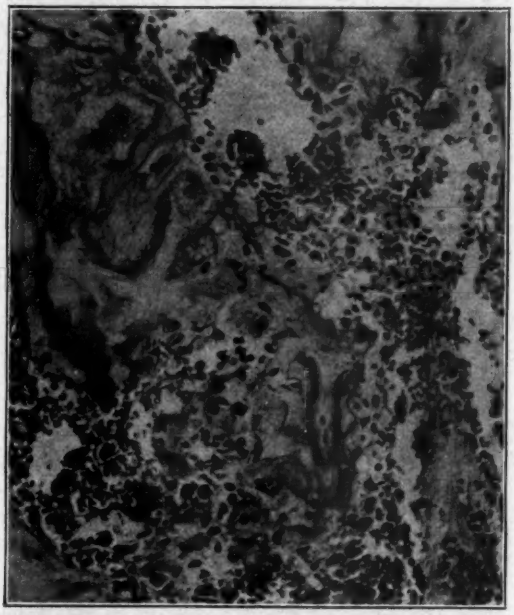

Fig. 15.—Detail of Figure 13, starvation group, animal 9; segment of bone in marrow space showing predominance of resorption lacunae, osteoclasts and fragmentation and dissolution of bone matrix. (Hematoxylin and eosin; ×320.)

In the normal diet group, the destructive changes in the interdental septum were limited to the bone margin below the inflammatory cells. In the starvation group, in addition to the marginal bone changes, described in the normal diet group, the lacunae normally containing osteocytes were, for a considerable depth in the septum, enlarged and devoid of osteocytes and the bone matrix was markedly granular. In the normal group, marginal resorption was evidenced only by irregular concavities. In the starvation group, on the other hand, there was fragmentation and dissolution of the peripheral bone matrix in addition to irregular concavities of resorption. Bone formation was observed adjacent to the resorption lacunae in the normal diet group. Such bone formation was absent in the starvation group. The extent of bone loss in the normal group in a thirty-day period was exceeded by

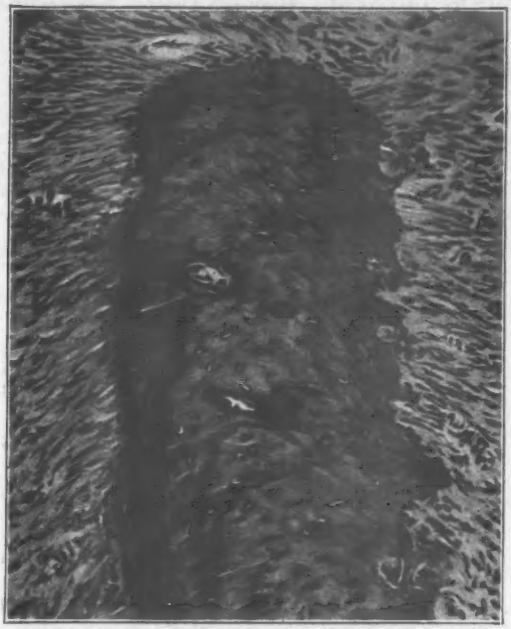

Fig. 16.—Normal diet group, animal 7; interdental septum, in absence of inflammation, showing resorption lacunae and osteoclasts on right (mesial), pale new bone matrix with osteoblasts at left (distal) and border of new bone matrix at gingival crest. (Hematoxylin and eosin; ×160.)

Fig. 17.—Starvation group, animal 23; interdental septum, in absence of inflammation, showing resorption lacunae at right (mesial) and peripheral fragmentation of bone matrix at left (distal) and along gingival crest. (Hematoxylin and eosin; ×150.)

that which occurred in an average eight-day period in the starvation group. The combined osteocyte and bone matrix changes in the starvation group resulted in necrosis of segments of the interdental bone, which became segregated from the underlying septum and underwent fragmentation and dissolution, and thereby effected a marked reduction in the height of the septum.

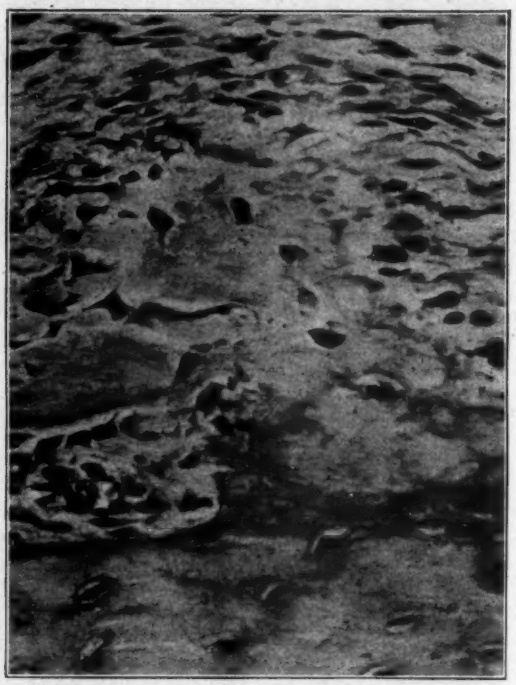

Fig. 18.—Detail of Figure 17, starvation group, animal 23; gingival crest of interdental septum showing fragmentation of bone matrix and enlarged lacunae empty of osteocytes. (Hematoxylin and eosin; ×560.)

GENERAL COMMENT

Demonstration of the fact that bone loss subjacent to gingival inflammation is regulated by systemic influences introduces a significant basic concept into the field of periodontology. Unless the implications of this concept are fully recognized, progress in research or advances in treatment are unlikely.

We have demonstrated the reality of the participation of systemic influences in effecting the bone loss observed subjacent to gingival inflammation at necropsy or in the clinic. This makes the interpretation of such bone loss as a purely "local type" of change a questionable procedure. The truth of this is apparent from our experiments, which reveal that the response of the periodontal bone to gingival inflammation is an expression of the prevailing status of the entire skeletal system. In both the normal diet group and the starvation group, the prevailing status of the entire

Fig. 19.—Starvation group, animal 28; interdental septum, in absence of inflammation, showing resorption lacunae at left (mesial) and fuzzy remnant of pale pink matrix at right (distal) which blends with adjacent connective tissue. The gingival crest presents fragmentation of the bone matrix with loss of substance. (Hematoxylin and eosin; ×80.)

Fig. 20.—Detail of Figure 19, starvation group, animal 28; gingival crest of the interdental septum, in absence of inflammation, showing fragmentation and dissolution of bone matrix and enlarged lacunae empty of osteocytes. (Hematoxylin and eosin; ×720.)

skeletal system is the "bone factor" which regulates the severity of the bone loss in both the presence and the absence of gingival inflammation. The greatest hope for progress in periodontal treatment lies in the regulation of the "bone factor" in favor of the individual in order to minimize the destructive influence of local inflammation. The importance of clarification of this "bone factor" suggests the direction for future research in periodontology.

Such an approach to the treatment of periodontoclasia does not detract from the importance of continued improvement in the methods employed for eliminating the local inflammation which has been demonstrated as capable of causing periodontal bone loss. However, since such bone loss is regulated by a "bone factor" of systemic origin, and since both clinical evidence and the findings here have substantiated the occurrence of periodontal bone loss in the absence of inflammation, the comparative possibilities of local and systemic

Fig. 21.—Normal diet group, animal 6; interdental septum subjacent to area of artificially induced inflammation, showing infiltration of lymphocytes in relation to bone margin. (The localized accumulation of polymorphonuclear leukocytes immediately below the gingival epithelium is a reaction to the presence of a small bone fragment.) (Hematoxylin and eosin; ×98.)

therapy in attaining the ultimate control of periodontoclasia are apparent.

SUMMARY

A series of animal experiments were conducted to determine what effects an alteration of the systemic background would bring about in the bone subjacent to areas of gingival inflammation. The following conclusions were established by the findings:

I. The bone of the jaws undergoes changes similar to those which occur in other bones as the result of generalized skeletal disturbances of systemic origin.

II. In the absence of gingival inflammation, bone loss may occur in periodontoclasia as a result of generalized skeletal disturbances.

III. Inflammation in the gingival crevice may result in bone resorption and reduction in the height of the interdental septa in the absence of generalized skeletal disturbance.

IV. The severity of bone loss subjacent to gingival inflammation is increased by generalized skeletal disturbances of systemic origin.

It is suggested that since a "bone factor" of systemic origin regulates the progress and severity of bone loss in periodontoclasia, both in the presence and in the absence of gingival inflammation, research in the field should be directed toward determining the nature of this "bone factor."

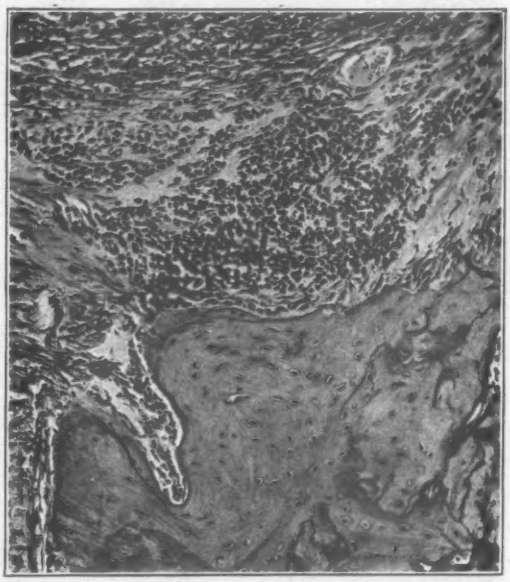

Fig. 22.—Detail of Figure 21, normal diet group, animal 6; margin of interdental septum subjacent to area of artificially induced inflammation showing area of lacunar resorption at right adjacent to new bone matrix lined with layer of osteoblasts at left. The underlying bone is unaltered. (Hematoxylin and eosin; ×180.)

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Fig. 23.—Starvation group, animal 9; interdental septum subjacent to area of artificially induced inflammation showing chronic inflammation in connective tissue above bone margin. There is an absence of bone formation in relation to the entire septum. (Hematoxylin and eosin; ×90.)

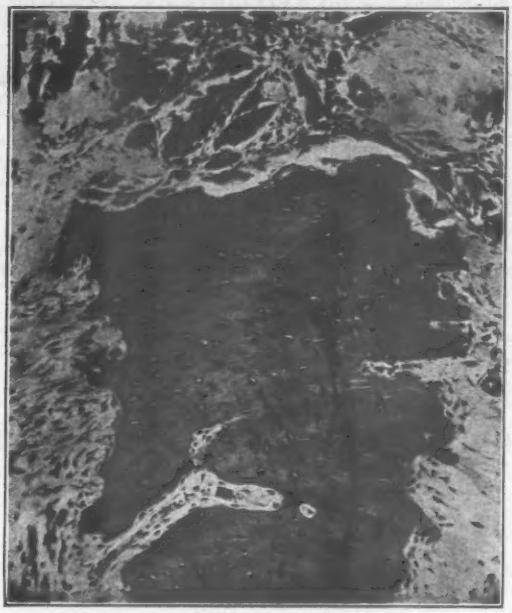

Fig. 24.—Detail of Figure 23, starvation group, animal 9; margin of interdental septum subjacent to area of artificially induced inflammation. The entire bone margin is formed by shallow concavities. For a considerable depth below the margin, the osteocyte lacunae are enlarged and empty. Fragments of bone are seen in the connective tissue. There is no evidence of bone formation. (Hematoxylin and eosin; ×150.)

Fig. 25.—Starvation group, animal 9; interdental septum subjacent to area of artificially induced inflammation showing separation of necrotic bone segment from remainder of septum. A row of multinuclear osteoclasts is seen between the two bone regions. (Hematoxylin and eosin; ×110.)

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APLASIA OF THE CROWN OF THE TOOTH; HEREDITARY HYPOPLASIA OF THE ENAMEL: REPORT OF TWO CASES

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APLASIA of the crown of a tooth is due largely to hypoplasia of the enamel. Few such anomalies have been reported. In aplasia of the crown of a tooth, roentgenographic examination is useful to determine the decreased size of the root canal. In treatment of almost every case, there are two alternatives: extraction and replacement by a denture or construction of porcelain jacket crowns to restore the teeth.

Colyer and Sprawson (1931) reported a case in which the surface of the crowns was a dark brown to amber yellow and was semitransparent. Olson (1938) saw a patient aged 20 years whose teeth presented a straw-colored, tobacco-stained appearance. Of the two cases that I observed, one had dark brown to amber yellow crowns. The other has teeth of bluish hue and semitransparent.

Contrary to the general belief, enamel hypoplasia *per se* is not an important etiologic factor in dental caries. It is true, however, that hypoplastic teeth decay more rapidly than normal teeth once decay has started. If a person is immune to caries, intact as well as hypoplastic teeth will remain free of caries. If he is susceptible, the teeth will decay regardless of whether they were intact or hypoplastic, but the destruction will spread more rapidly and extensively in hypoplastic teeth.

REPORT OF CASES

CASE 1.—*History.*—A man, aged 30, in good general health, gave a history, on the father's side, of hypoplasia of the enamel affecting the grandfather, father, aunt and uncle. His brother, aged 17, and daughter, aged 22 months were also affected. The grandmother, two aunts and uncle were unaffected.

This patient's father wore artificial dentures from the age of 40.

Oral Examination.—The oral mucosa and gingivae were normal. Sixteen teeth were present, all others having been extracted because of caries. All the lower teeth had been unable to withstand the forces of attrition and had become worn down to the gingival

margin. The remaining teeth appeared dark brown to amber yellow. Some of the teeth were whiter than others. Considerable protrusion of the mandible had resulted from the wearing down of the lower teeth.

Diagnosis.—Hereditary hypoplasia of the enamel was diagnosed.

Prognosis.—The prognosis was considered grave, since these teeth are always lost.

Treatment.—Extraction of the remaining sixteen teeth and construction of full dentures were advised, with particular attention to restoring the vertical dimension.

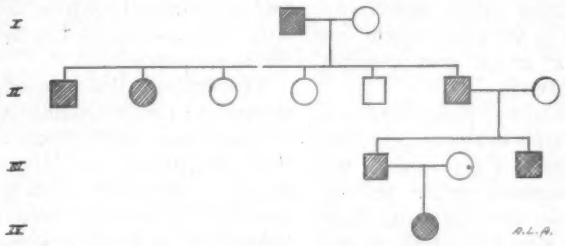
Comment.—The condition caused the patient much concern. He felt even greater anxiety about the outcome of his 22-month-old daughter's condition.

hue, having the so-called "dead" appearance, and were semitransparent. The teeth were not noticeably worn down.

Diagnosis.—Hereditary hypoplasia of the enamel was diagnosed.

Prognosis.—The prognosis was poor. The most that could be hoped for was that the primary teeth could be retained until the normal exfoliation age.

Treatment.—Porcelain jacket crowns were impossible and dentures were not advisable. If caries should become extensive, extraction might be necessary, but careful guidance and restorative work might preserve the primary teeth until exfoliation occurs. A low carbohydrate diet was considered as possibly indicated.



PEDIGREE SHOWING OCCURRENCE OF HEREDITARY APLASIA OF THE ENAMEL IN FOUR GENERATIONS. SHADED SYMBOLS INDICATE APLASIA. SQUARE SYMBOLS ARE FOR MEN AND CIRCULAR SYMBOLS ARE FOR WOMEN. ROMAN NUMERALS INDICATE GENERATIONS.

CASE 2.—*History.*—A girl, aged 22 months, in general good health, on the father's side of family had a great-grandfather, grandfather, great aunt, great uncle, uncle and father affected with hypoplasia of the enamel. The great-grandmother, two great aunts and a great uncle were unaffected.

Oral Examination.—The oral mucosa and gingivae were normal. The twenty teeth present were all fairly well formed and comparatively little affected by caries. The posterior teeth were all somewhat whiter than the anterior teeth. The teeth were of a bluish

Comment.—Grave psychologic problems may arise in the young patient's life owing to the unnatural appearance of her teeth. Unkind remarks of classmates may cause her to have a feeling of inferiority.

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BLOOD DYSCRASIAS AND THEIR RELATIONSHIP TO THE DENTIST*

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SYMPTOMS of blood dyscrasia of dental interest have been known for a long time. According to Carroll LaFleur Birch's[1] interesting historical data, fatal bleeding after rubbing of gums was observed as early as the eleventh century. Concerning lesions of the gums, Shapiro[2] goes even farther back, quoting from MacNevin-Vaughan's book to the effect that it was Hippocrates who noted gum changes, apparently in leukemia.

The last twenty-five years, however, brought more results in this respect than any previous period. The reason for this is partly developments in the field of hematology. I am thinking particularly of the last two decades, which, in the history of hematology, are marked by many important achievements. I should like to point out the introduction of liver treatment in pernicious anemia, which had an unprecedentedly outstanding effect on the entire field of hematology. In the light of liver treatment, we gained a better understanding of iron treatment and deficiency anemia. To mention just a few of the other developments, there are recognition of neutropenic state, including the different clinical forms of agranulocytosis; improved recognition of different clinical forms of acute leukemia and their subleukemic phases, especially through the aid of sternal puncture; the discovery of vitamin K and its importance in prothrombin formation; as well as many other developments in serology; the improved technic of plasma-blood transfusions and blood banks, which now, during the war, are of paramount importance. Some of these newly or better recognized types of blood dyscrasia, such as agranulocytosis and acute leukemia, are associated with the most marked tooth, mouth or throat symptoms.

Throughout this period, dentistry showed an ever-increasing interest in its systemic connections. It is natural, therefore, that, during the last two decades, tooth, mouth and throat symptoms of blood dyscrasia have been increasingly recognized and observations published in greater numbers from both dental and medical fields.

Since, in some types of blood dyscrasia, symptoms may appear initially in the mouth, a patient is apt to first seek the aid of the dentist. For prevention, the importance of early recognition of these symptoms is obvious, and it was emphasized in the comprehensive reviews of Aison,[3] Aubertine,[4] Cahn,[5] Cook,[6] Lefkowitz, Darlington and Brown,[7] Rosenthal[8] and a number of others. In other words, it is the emphasis on the fact that a sometimes insignificant-appearing lesion in the mouth is not merely indicative of a local condition, but is just one symptom of a usually severe systemic disease. With an exact clinical history, family history and laboratory examinations, correct diagnosis can be established and treatment be begun immediately.

Concerning dental therapy in blood

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dyscrasia, the most important problem confronting the dentist in every-day practice is tooth extraction. We may classify this into two groups: (1) tooth extraction with unrecognized blood disorders and (2) tooth extraction with properly diagnosed blood dyscrasia. Such a grouping again emphasizes the importance of recognition of the systemic nature of the disease. If dental treatment of a patient whose blood disorder has been properly diagnosed is necessary, treatment is carried out at an optimum time, under optimum circumstances and with a carefully selected technic, and with a well-prepared patient. This is usually the result of cooperation among dentist, physician and clinical pathologist. Such teamwork is well rewarded in beneficial results. Dental treatment of patients with unrecognized blood dyscrasia is work in the dark, with frightening results.

Among the types of blood dyscrasia of importance for the dentist, I should like to mention some examples of anemia. First, I would like to say a few words concerning the normal cellular composition of the blood. One cubic millimeter contains 4,500,000-5,000,000 red cells, 4,000-10,000 white cells and about 250,000-500,000 thrombocytes. On the stained blood smear, the white cells appear in the following proportions: 2-3 per cent stabs, 58-62 per cent polymorphonuclear leukocytes, 2-5 per cent eosinophils, 2 per cent monocytes and 20-30 per cent lymphocytes. While the entire series of cellular development is present in the bone marrow, spleen and lymph nodes, respectively, normally only the fully mature cells appear in the peripheral circulation. At present, we know relatively little of the manifold intricate mechanisms by which their maturation and delivery are governed and I cannot here go into the many observations and experiments which have led to different interesting theories. I should like to mention the fact that a certain number of all these cells are necessary for maintenance of normal life. If the number of red blood cells decreases to below 4,000,000, the condition is known as anemia. A reduction in the number of platelets below 40,000 causes purpuric bleeding. Clinical observations indicate that, among the white blood cells, the neutrophilic granulocytes also have a "critical level" (which is thought to be about 800-1,000 in one cubic millimeter in absolute figures), and if the number of neutrophilic leukocytes drops below this level, clinical symptoms of severe neutropenia appear which are well differentiated from agranulocytosis.

Instead of going into the classification of anemia, we should mention only those types that are frequently associated with mouth symptoms. In any type of severe anemia, there may be pallor of lips and mucous membranes of the mouth. In one type of anemia, glossitis occurs frequently. Among these, the tongue symptoms of pernicious anemia are well known. Hypersensitivity and tenderness may be among the earliest symptoms, sometimes long preceding the blood changes. Slowly the typical glossitis develops. The inflamed tongue is not coated, but is red, raw and bulky, owing to the damaged covering epithelium, inflammatory hyperemia, edema and infiltration with inflammatory cells. The changes are marked at the tip and sides of the tongue. As a result of the inflammation, superficial vesicle formation and small ulcers also may be present, though rarely. All these subjective and objective symptoms show fluctuations in intensity. Relief and aggravation alternate. Eventually, the inflammation passes into the chronic, atrophic stage. The tongue becomes pale, glazed and smooth, from loss of the papillae.

Stomatitis is, in these cases, very rare. Usually, the inflammation is confined to the tongue and, to a lesser degree, to the pharynx. Other parts of the oral mucous membranes are pale or normally colored,

even if the glossitis is acute, in striking contrast to the bright "beefy" tongue. We do not know of any characteristic pathologic change in the gingivae. However, it is asserted that pyorrhea is common. Since the typical symptoms of pernicious anemia appear at about middle age, this is difficult to judge. Ordinary tooth diseases seem to be frequent, and also an early loss of teeth.

At present, we know that the described changes in the tongue are not confined to the clinical picture of pernicious anemia. They are frequently present in other types of macrocytic anemia such as *Diphyllobothrium latum* infection, the macrocytic anemia of pregnancy, gastric carcinoma, cirrhosis of the liver, complete and, in some cases, partial gastrectomy and intestinal obstruction. The tongue symptoms are also frequently found in microcytic anemia, such as idiopathic hypochromic anemia, the Plummer-Vinson syndrome and other types of hypochromic microcytic anemia. In microcytic anemia, the patient usually shows characteristic symptoms of chronic atrophic glossitis without being able to recall any symptoms of an acute start.

Obviously, tongue symptoms are independent of the macrocytic or microcytic type of anemia. It is interesting to note, however, that they are confined to the group of "deficiency anemias." Experimental data, to mention only the names Miller and Rhoads,[9] Hutter, Middleton and Steenbock,[10] seem to show that glossitis is of nutritional origin. It is caused by a deficiency, as in pernicious anemia. Castle[11] defined it as ". . . resulting not by a direct inadequacy of the diet, but from a conditioned deficiency produced by the failure of some functions of the normal stomach to take place in the stomach of the patient with pernicious anemia . . ." The substance, lack of which is causing the tongue symptoms, probably belongs to the group of vitamin B complex. That it is not the same as that causing the anemia is clear from the following facts: 1. The severity of the anemia does not parallel the severity of tongue symptoms. 2. It is independent of the type of anemia, which may be macrocytic or microcytic. 3. In case of tongue symptoms in iron deficiency anemia, the condition can be corrected by administration of iron. The glossitis can be improved by treatment with crude liver extract. 4. The anemia and glossitis are not closely linked and the glossitis is the result of nutritional deficiency and not the anemia. This is also evident from the fact that glossitis is absent in large groups of other anemia types, such as many of the hemolytic types.

The effect of crude liver extract treatment on the tongue symptoms must be emphasized. In pernicious anemia, it brings about a definite improvement, with return to normal and reestablishment of the atrophic papillae. The effect is similarly obvious in the other mentioned types of anemia and other conditions. Administration of nicotinic acid may enhance the improvement. (Jolliffe and Stern.[12])

It is obvious that dental treatment, such as tooth extraction in pernicious anemia, should be carried out with close observation of clinical symptoms and frequent checking of the blood. Fink[13] reported two cases wherein extraction of teeth in pernicious anemia resulted in death. It seems, however, that, with proper precautions, if the patient is adequately treated with liver, normally no difficulties should be encountered. One of my patients, a woman aged 78, in whom pernicious anemia became apparent eighteen years ago, belongs to the group of the first pernicious anemia patients whose lives were saved by the discovery of liver treatment. She had five teeth extracted six months ago and a denture replaced. There was no change noticeable in the normal red cell count after the extractions and no change in her usual liver dosage was necessary.

In microcytic hypochromic anemia, there is usually pallor of the lips, tongue

and oral mucosa. The possible change of the tongue, chronic atrophic glossitis, was mentioned before. Causes of this anemia are numerous: low hemoglobin content and low red cell count are mainly one symptom of some disease which is to be treated. If the anemia is marked, it is frequently associated with a bleeding tendency, mostly due to a lowered platelet count. The decreased resistance of the patient may cause considerable difficulties. As Thoma[14] warns: ". . . there is a decided predisposition to infection and ulceration," and "septicemia has been reported to occur following the extraction of infected tooth." If there is no urgency, the patient may be treated concentratedly with iron and crude liver extract for several weeks before any dental work is performed. With a normal red cell count and normal hemoglobin content, success of the dental work is, of course, greatly insured. In case of urgency, repeated blood transfusion may be necessary.

"Aplastic anemia" is today applied to a rather heterogenous group of diseases which result in aplasia of the bone marrow leading to progressive anemia, leukopenia and thrombocytopenia. It may occur as a disease of unknown etiology, called "idiopathic aplastic anemia," or as a condition produced by ingestion of certain poisonous chemicals (benzene, arsenic), infections or x-ray or radium contact, or may be associated with other diseases such as kidney, liver or endocrine organ conditions. Frequently repeated transfusion is the only possible means to lengthen the life of such patients, who usually die in from a few days to a few months after the appearance of symptoms. Mouth symptoms are rare, occurring at a later stage of the disease when anemia is already marked. At that time, extreme pallor of the skin and mucous membranes is the most apparent symptom. C. P. Rhoads[15] found acute ulcerative stomatitis and glossitis in a number of such patients. In some of these cases, he also found atrophy of the papillae of the tongue, but these changes are slight and localized and not similar to papillary atrophy of the tongue as in pernicious anemia.

After anemia, I should like to mention a condition in which the red cell count is increased above normal: polycythemia vera. There is normally a marked bluish red cyanotic color of the lips, tongue and gingivae. It is interesting that in some instances the condition ends in leukemia. Prolonged hemorrhage may occur after extraction. This may be of benefit to the patient, at least for a short time, substituting for the beneficial effect of venisection and lowering the blood volume, red cell count and hemoglobin toward the normal level.

I can discuss only briefly the hemorrhagic diseases of interest to the dentist, in spite of the fact that bleeding is the most common symptom of blood dyscrasia in the mouth cavity. Bleeding may be the sign of a primary hemorrhagic disorder, but it may also introduce or accompany many disorders of red or white blood cell formation. It is a property of normal blood to remain fluid while circulating within the vessels, but it clots if, through any injury, the blood is shed. In injury, from the injured tissue, mainly from disintegrated platelets, a substance, thromboplastin (thrombokinase, cephalin), is liberated which initiates the rather complex mechanism of clotting. The number of platelets is normally 250,000-500,000. If it is lower, below 50,000, or if the platelets are qualitatively abnormal, purpuric bleeding usually sets in.

In our present classification, one large group of bleeding diseases is characterized by an abnormally low number of platelets in the circulating blood. Besides the decreased number of platelets, there is always capillary damage which, through recent research, gains increasing significance. The condition may appear as "essential" thrombocytopenic purpura

(purpura hemorrhagica, or Werlhof's disease), in which, besides the quick, but temporary relief of blood transfusions, irradiation of the usually enlarged spleen or splenectomy brings about an increase in the number of platelets and permanent remission. It may also appear as "symptomatic" thrombocytopenia, when the decreased platelet count is the sequence to some other condition, such as infections or the effect of certain chemical or physical conditions; or it complicates other types of blood dyscrasia such as pernicious anemia, severe mycrocytic, hypochromic anemia; leukemia, and aplastic anemia.

The second group of hemorrhagic diseases is the group of so-called "non-thrombocytopenic" purpura, in which the platelet count is normal and the bleeding symptoms are due to some damage of the capillaries. This type of purpura appears in association with various conditions, such as infectious conditions and avitaminoses. The allergic type of purpura is impressive.

Both these groups cause mostly spontaneous "purpura"; that is, small, pinpoint-like bleeding areas (petechiae) or slightly larger areas (ecchymoses) in the skin or mucous membranes covering small or larger areas. They may, however, cause diffuse oozing of blood from the gums, severe nosebleed and spontaneous diffuse bleeding from other organs. Also, bleeding may not be spontaneous, but may follow such injuries as tooth extraction. Repeated bleeding in these diseases may lead to anemia.

Differential diagnosis between thrombocytopenic and non-thrombocytopenic purpura is based on laboratory and clinical findings. Since the capillaries are pathologically changed in both groups, the capillary resistance test (tourniquet test) is positive in both groups. While in non-thrombocytopenic purpura, the bleeding and clot-retraction time are normal, bleeding and clot-retraction time in thrombocytopenic purpura are prolonged in varying degrees. Clinically, the fact that non-thrombocytopenic purpura shows certain allergic characteristics besides bleeding, such as erythemia, urticarial wheel formation, angioneurotic edema, etc., may help in the differential diagnosis. Inflammatory symptoms of the joints (Schönlein type) or abdominal symptoms (Henoch type) may also be present.

It should be emphasized from the dentist's viewpoint that, in these diseases, both the clinical features and the laboratory reports show definite fluctuations. Bleeding symptoms in essential thrombocytopenic purpura may be absent for long intervals, only to disappear and reappear.

Laboratory findings may be within normal range for long periods. It is therefore of importance that the dentist take a careful clinical history, which may show whether the patient has had repeated bleeding symptoms from early childhood. Also, in any suspicion of such hemorrhagic disorders, laboratory examinations should be made several times, at different intervals. The result of a single laboratory examination is not sufficient.

The rôle of infections should be especially emphasized in connection with hemorrhagic disease. Infection or dental disease as a focus of infection may depress the function of the bone marrow and the decreased platelet count lead to bleeding. Bleeding and consecutive anemia may further decrease the resistance of the patient and aggravate the infection. Thus, a vicious cycle is formed which, if the gravity of the situation is not realized in time, may have serious consequences. If infected teeth are the foci of infection, they must be removed, but timing of such an operation and preparation of the patient are of paramount importance. If the patient is suffering from an acute stage of the infection, with superimposed bleeding tendencies, the outcome of a hurried operation

may be fatal. If the operation is preceded by blood transfusion and proper medical treatment, the outcome, with a normalized bleeding mechanism, may prove more favorable.

Hemophilia, a hereditary disease, is sex-linked in its transmission. Females transmit the disease, symptoms of which occur only in males. It is believed that undue stability of the platelets causes the disease. However, recent experiments suggest that the plasma may be responsible for the defective clotting mechanism. Because of the hereditary nature, in tracing the family history, the dentist should inquire carefully concerning bleeding of members of the patient's family. Positive replies may arouse suspicion of hemophilia. In this disease, excessive bleeding, which may appear spontaneously or on injury, is characteristic. Bleeding, which may appear early in life, shows great fluctuation in the tendency to bleed. The eruption and loss of teeth are usually sources of severe, sometimes fatal bleeding. In Carroll Birch's[1] report on ninety-eight cases, in twenty-five cases of fatal hemorrhage, six followed tooth extraction. Nineteen of these patients had their first hemorrhage from the mouth. Ninety-four had hemorrhages sooner or later from the gums and tongue.

In the laboratory examination, the more or less prolonged clotting time is diagnostic, with other findings negative. It is well known and well demonstrated in Birch's graphs that the clotting time of one's blood varies at markedly different intervals. At times, it is prolonged; at others, it may be close to normal. If any dental work must be done, study of the clotting time should precede it, since, obviously, proper timing is of the utmost importance. Tooth extraction should be preceded and followed by frequent blood transfusion. Many cases were reported in which, by aid of transfusion, absolute rest and careful local treatment, tooth extraction was successfully carried out. As to the technic of tooth extraction, Birch and Snider[16] achieved good results by using the method of placing rubber bands around the teeth. By this constant pressure, vessels close slowly and denudation and expulsion of the teeth follow without undue bleeding. Another method is that of Dalitsch,[17] who applies, after extraction, a close-fitting molded splint or vulcanite covering the site of injury. In the local treatment of such patients, it seems important that no undue packing and pressure be exerted since bleeding may spread deeply into the tissue spaces. In one of Balyeat's[18] cases, death was caused by asphyxiation due to pressure on the trachea.

Walker[19] emphasized the proper diagnosis in case of hemorrhage. Sometimes, an extensive but only traumatic hemorrhage may lead the dentist to believe that his patient is a bleeder. On the other hand, Birch's excellent examinations showed that symptoms of real hemophilia may be of a very mild degree. To establish the diagnosis is of extreme importance.

In all these cases, a few minutes taken to delve into family and clinical history may be well rewarded. This, together with the necessary laboratory examinations, may not only postpone extraction, but also save the patient's life—and the dentist's reputation.

It is certainly difficult for the dentist in routine practice to obtain these data while working on large numbers of patients, and even those who collect such data in routine manner are apt to become neglectful after a time because years may pass without their being confronted with a case of hidden blood dyscrasia. However, dentists who have had such experiences are aware of the value and importance of collecting such data as a matter of routine. Tooth extraction in such cases may elicit a series of stormy symptoms, sometimes ending fatally. To quote Locher[20]: "Here let me say, be sure of your patient; obtain

a complete history. This is very essential, for it is the ninety-ninth case out of a hundred that will bring you grief."

I cannot here go into the interesting discoveries concerning vitamin K and its effect on the clotting mechanism or other vitamin deficiencies associated with bleeding symptoms, nor can I consider the subject of glandular fever or other conditions frequently associated with mouth symptoms. Instead I should prefer to discuss briefly agranulocytosis and acute leukemia, which are of particular interest from the dentist's point of view because their earliest symptoms frequently arise in the mouth cavity. Early diagnosis in acute leukemia may somewhat prolong the life of a patient, and in agranulocytosis may even save it.

Agranulocytosis was first described under that name by Schultz in 1922. The course of acute "fulminating" agranulocytosis is short and serious. The onset is sudden, usually with sore throat, toothache or stomatitis, causing excruciating pain, with necrotic ulcers developing quickly on the tonsil, gums or elsewhere in the oral cavity. The necrosis may extend deeply into the tissues producing intolerable pain and fetor oris. The absence of any inflammatory reaction, a necrotic character and lack of pus formation are the outstanding features of these lesions. The necrotic ulcer may be covered with a dirty yellowish gray membrane. This or the surface of the ulcer contains many Vincent's organisms. The patients are feverish and may have jaundice. The spleen and liver may be enlarged. The white cell count shows a profound decrease, with leukopenia and neutropenia.

If not treated, patients with this acute condition usually die within from five to seven days. The subacute and recurring forms are more prolonged and may be complicated with secondary infections. The chronic forms may last for years or even decades.

Necrotic ulcers may appear not only in the mouth cavity, but also anywhere in the system. Probably, they are directly related to the low number of neutrophils in the circulating blood. In a frequently quoted publication, Roberts and Kracke[21] observed a case of relapsing agranulocytosis in which a marked decrease in the number of neutrophilic polymorphonuclears definitely preceded the appearance of mouth lesions. This has frequently been confirmed in the literature.

The presence of a certain number of fully matured white blood cells, neutrophilic polymorphonuclears, leukocytes, are necessary to life. There is clinical and experimental evidence that life cannot be maintained more than six or seven days without these cells. It is also stated that when the number of neutrophilic polymorphonuclears drops below a given number—approximately 800 per cubic millimeters in absolute figures—symptoms of agranulocytosis appear immediately. Why neutrophilic polymorphonuclears are of such importance to the organism is not yet understood. Since Metchnikoff, we know that they may act as a barrier against invading microorganisms. They play an important part in producing immune bodies and enzymes, and perhaps also in the oxydative processes of the tissues, but all this does not fully explain their extreme importance. Why lesions usually appear first in the oral cavity is still a matter of dispute. The cause may be complex. A decrease of tissue oxydation in the oral mucous membranes, an absence of normal neutrophilic defense in the circulating blood, in the tissues and in the saliva in which neutrophils are normally found in large numbers, friction caused by chewing and swallowing, all may predispose the mouth cavity to lesions, so that all these factors may play a part. The large number of micro-organisms present in the oral cavity are ineffective in the normal defense, but may cause severe secondary infection when this de-

fense is weakened. The rôle of the number of neutrophils in the control of Vincent spirochetes in the gingival crevices was studied by Kitchin[22] and the importance of mouth hygiene as regards the appearance of necrotic lesions was emphasized by Kaiser.[23]

On the other hand, we cannot omit the contradictory results of certain animal experiments. Miller and Rhoads[24] noted the development of ulcerative stomatitis and severe leukopenia in dogs kept on a black-tongue producing (vitamin B deficient) diet. The decrease of polymorphonuclears in these animals followed, and did not precede, development of mouth lesions. Syverton and Lawrence and their associates[25] studied the "infectious feline agranulocytosis," a virus disease of cats, which is associated with profound granulocytopenia and less marked leukopenia. Oral lesions were not noted. It is questionable, however, how far these experiments can be applied to human agranulocytosis.

As to the etiology of agranulocytosis, a certain hypersensitivity or latent disorder in the white cell formation must be supposed. The disease may be initiated by several conditions, the most important of which is amidopyrine therapy. It is to Kracke's credit that the attention of American physicians and dentists was focused on the danger of amidopyrine therapy and thus the incidence of the disease decreased. It was also Kracke who anticipated that sulfanilamide would take over the rôle of amidopyrine. The disease may be elicited by administration of several other drugs, such as heavy metal (gold, silver) and arsphenamine.

In treatment of agranulocytosis, we must first eliminate all drugs, and fight against infection. The use of nucleic acid derivatives, liver extract therapy and x-ray radiation in small doses to the long bones may be of help, but the best remedy seems to be blood transfusion. The depressed function of the bone marrow may improve under proper treatment and there is always a chance for recovery. I refer, of course, to cases in which early diagnosis and early treatment may actually save the patient's life.

The rôle of teeth in this disease deserves special attention. There are many published reports of tooth extraction followed by fatal agranulocytosis. I refer to Hill's[26] paper published in 1926 in which he noted the close and probably direct connection of fatal agranulocytosis with tooth extraction immediately preceding it.

As to the literature, I wish also to mention the comprehensive papers of Appleton,[27] Cook,[6] Schaeffer[28] and many others. There are, however, many published cases of persons who survived agranulocytosis initiated by tooth extraction and, in some instances, not only survived the agranulocytosis, but also recovered entirely and were cured of the disease. Nevertheless, there are some reported cases of recurring attacks of agranulocytosis after extraction of infected teeth, with freedom from the disease and no recurrence of the attacks. I have also observed cases which strongly supported the view that in agranulocytosis, recovery may follow removal of infected teeth acting as foci. Dennis[29] emphasized the significance of infectious foci in agranulocytosis. Because of an insufficient number of cases, the problem is still a matter of dispute. I believe that, in every case of agranulocytosis, the possible etiologic rôle of focal infection should be considered just as carefully as we investigate, as a matter of routine, any use of amidopyrine or other drugs or other factors. Obviously, whenever there is reason to believe that dental infection may be the cause of the disease, the teeth affected should be removed with necessary precautions.

Among the most important precautions to follow is the sound advice of Coolidge[30] in such cases: "No dental operation should be performed, espe-

cially no operation that causes injury to the tissue, until after the medical treatment has improved the blood picture." Experience has shown that tooth extraction in patients suffering from agranulocytosis is inevitably followed by a drop in number of neutrophils.

The depressing effect of tooth extraction on the white cell count in agranulocytosis is certainly an interesting problem for the dentist. Ralph L. Ferguson and I studied the effect of tooth extraction on the white cell count on albino rats and rabbits and found that tooth extraction in itself, regardless of the effect of the anesthetic, had an immediate effect on the white cell count. This effect varies in animals with normal or with experimentally stimulated or depressed function of the bone marrow. In normal animals, the tooth extraction causes a transient leukopenia, followed by leukocytosis. In animals with stimulated bone marrow function, the response is leukocytosis only. In animals with a depressed function of the bone marrow, the immediate reaction was an even deeper depression of the white cell count. It should be pointed out that tooth extraction in normal animals did differ in its effect on the white cell count from other operations (skin incision; laparotomy). Our results, in the form of a lecture, were published in December 1940.[31] We do not know how far these results can be applied to human beings. So far, the results have been confirmed on human material by P. S. Faillo,[32] in 1942.

Among other diseases of white cell formation, acute and chronic leukemia should be mentioned. In discussing mouth symptoms, I wish to say that acute and chronic leukemia is frequently discussed together in the literature and referred to as "leukemias." Acute leukemia differs from chronic leukemia sharply in the clinical course and blood findings as well as in the mouth symptoms.

In chronic lymphatic or myelogenous leukemia, mouth symptoms are relatively rare. Spontaneous bleeding of the gums, severe nosebleed or undue and prolonged bleeding after slight injuries or tooth extraction may occur, indicating a disturbance of the bleeding mechanism; also spontaneous purpura covering the skin and mucous membranes may be one of the early symptoms which may clear up entirely later. Bleeding may be an early or introductory symptom, or may occur at any stage of the disease. Other early characteristic mouth symptoms are few.

Frequently, in cases of chronic lymphatic leukemia, tonsils are enlarged at a later stage in the disease as part of the general lymphatic hyperplasia. In the late stages also, pallor of the mucous membranes becomes more apparent with the increasing anemia. Normally, at this late stage, when the number of myeloblasts or lymphoblasts, respectively, in the blood increases, infiltration of the gums develops. In lymphatic leukemia, the infiltration prevails in places normally rich in lymphatic tissue; i.e., pharyngeal and sublingual regions. The infiltration with myelogenous cells is more pronounced in the gums. Stomatitis, ulceration and necrosis may occur in this stage, though it is relatively rarely. Kirshbaum and Preuss[33] indexed 123 cases of leukemia: 61 per cent (75 cases) acute and 39 per cent (48 cases) of the chronic type. In seven of the latter cases, acute inflammation and ulcerous processes in the oral cavity or throat developed.

In chronic forms of leukemia, oral symptoms are absent for many years, and, during these periods, dental manipulations may be carried out with relative safety. If tooth extraction is necessary, it is important to check the general condition of the patient. Careful examination of the blood is also necessary to determine the degree of anemia and the number of white blood cells and thrombocytes, and to check the coagulation and bleeding time. In moderate anemia, with normal bleeding and co-

agulation time and a white cell count between 50,000 and 80,000, I had teeth extracted in cases of both chronic myelogenous and lymphatic leukemia, with the result that the extraction as well as the healing was uneventful. Extraction was preceded and followed by frequent blood counts: before extraction, every hour for four hours immediately after extraction and daily for four successive days. There were no significant fluctuations in the white cell count. In one of our patients who was treated for chronic lymphatic leukemia for four years at the clinics of Loyola University, we observed that a lower left third molar tooth erupted after an unsuccessful operation. The blood count and general condition were not influenced at all. Naturally, the precautions mentioned cannot be neglected.

All this indicates that, in chronic leukemia, mouth symptoms are usually mild and generally develop only at about the end of the disease. In sharp contrast to this, the earliest symptoms of acute leukemia are oral and very severe in the majority of cases. These symptoms are: (1) bleeding from the gums or nose spontaneously, uncontrollable hemorrhage after slight trauma from the gums, and sometimes purpura; (2) ulcerations of the gums or any place in the oral and pharyngeal cavity including tonsils and tongue, frequently of necrotic character, resembling lesions of agranulocytosis; (3) infiltration of the gums, pharynx or laryngeal region with leukemic cells, and (4) stomatitis accompanying these lesions.

In a limited review like this, it may be justifiable to speak of "acute leukemias" generally. However, it is necessary to emphasize that there are many scattered and collected observations indicating that it is a mistake to refer to "acute leukemias" vaguely. While these different kinds of leukemia have certain similar features and characteristics, there are certain differences, not only in the blood picture, but in the clinical aspects as well. It is important, therefore, to attempt to trace these finer distinctions. Forkner[34] drew attention to these differences in the mouth symptoms. According to him, mouth lesions of acute monocytic leukemia are characterized by a marked swelling of the mucous membranes, mainly of the gingivae, frequently accompanied by ulceration and necrosis. These were absent in acute lymphatic and myelogenous leukemia, which had different types of symptoms, mostly diffuse cellulitis, an acute inflammation extending into the deeper tissues of the face. This view of Forkner's has not yet been universally accepted.

According to Kaufmann and Lowenstein,[35] "there is no clinical-pathologic picture which is characteristic of the cell type involved in acute leukoses." It is to the credit of Forkner that he was the first to recognize the possibility of using mouth symptoms for diagnosis of different types of acute leukemia. A search of the literature, as well as my own experience (nineteen cases of acute leukemia, three of which were monocytic and sixteen myeloblastic), convinced me that no other type of acute leukemia causes swelling of the gums to the extent that acute monocytic leukemia does, resulting in loosening and loss of teeth. On the other hand, infiltration on a smaller scale and necrotic ulceration were present in a large number of myeloblastic cases, especially those with a subleukemic start, and the white cell count started to rise only several days after the appearance of mouth lesions.

It seems quite possible that necrotic ulceration in both diseases—agranulocytosis and the acute subleukemic start of myeloblastic leukemia—is very similar, as it is caused by the same condition, lack of neutrophils. Swelling of the gums was also present in some cases, but only to a moderate degree. Since the swelling, according to the few histologic reports published thus far, is due to infiltration by the abnormal cells circulat-

ing in the blood, it seems logical to suppose that the monoblast of acute monocytic leukemia has a greater motility and infiltrating power than the myeloblast or lymphoblast. A study of the type and rapidity of movement of these cells explains this phenomenon to a certain extent. Since the type of abnormal cell in question has a direct and important influence on the infiltration, an analysis of these cases of acute leukemia may result in disclosing a close association between the characteristic mouth lesions and the type of cell in question. The handicaps are obvious in consideration of the fact that sometimes it is difficult to exactly define the pathologic cell. Such handicaps are present even in the case of the normal monocyte, as was pointed out by Kracke.[36] They are still considerably greater in the case of abnormal cells.

Our treatment of acute leukemia is very inadequate. Repeated blood transfusion may lengthen the life of a patient. Morphine is a great help in relieving the great pains and dyspnea. In these cases, no dental work should be done. Extraction of teeth may be quickly followed by death. This may be due to the fact that all the affected surfaces become necrotic, thus again an avenue is opened for invading micro-organisms. Toothache may indicate the beginning of this disease, being followed by rapid development of severe symptoms, whether dental work is done or not.

In the medical and dental literature, we read descriptions of typical, fully developed dental, mouth or throat symptoms. Recognizing the unusual is not difficult when symptoms are alarming or marked. The real pitfalls for the dentist lie in those cases of blood dyscrasia in which dental or oral symptoms and complaints are mild, simulating simple and common types of dental diseases. A severe toothache may mark the start of the clinical course in agranulocytosis or acute leukemia. Frequently, however, in such cases, there is an incongruity between severe complaints and negative local findings. The simple physical examination of the teeth and mouth alone does not help, even if the findings indicate a poor dental condition, inasmuch as patients with latent and heretofore unrecognized blood dyscrasia may also have an incidental pathologic condition of the teeth. The family must be made aware that the patient is suffering from a disease which is inevitably fatal, at least at present.

It is difficult to present in so brief a time a complete review of the numerous links between dentistry and hematology. However, I hope that I have indicated how important these associations are from a scientific as well as practical viewpoint. The scientific part is still in a phase of early development, requiring more clinical experience and further experiments and, what is more, increased interest in this subject. It would seem desirable, from a practical aspect, besides taking a clinical and a family history, to have routine blood examinations made in all cases in dental clinics in addition to the urine examination and Kahn test, just as is being done in all the great dispensaries. A routine determination of the white cell count, and bleeding and coagulation time, requires but a few minutes and sometimes may actually save a patient's life as well as assure the dentist that all possible precautionary measures have been taken.

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DEVELOPMENT OF THE OCCLUSION OF THE TEETH

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THE most essential function of man is respiration. Without sufficient oxygen, life cannot exist. The next most vital requirement is nourishment, for the building of the body and its continued maintenance. According to Nature's plan, food is taken into the mouth, where it should be properly masticated and mixed with saliva, before continuing on its course through the digestive tract. Normal mastication is necessary in preparing food for the maximum benefits of digestion and assimilation, and it is therefore essential to normal growth and health. Normal function of the chewing mechanism is possible in the highest degree only if there is a normal arrangement of the teeth—normal occlusion. I shall discuss the development of occlusion of the teeth from the viewpoint of the clinician.

We all know that at birth there is considerable development of the crowns of the twenty deciduous teeth, and that the jaws also contain the germs of thirty-two permanent teeth. The deciduous teeth begin to erupt at from 6 to 9 months, and usually the dentition is complete at 2 years of age. The roots of these teeth continue to grow and usually are fully developed when the child is 3 years old.

The development of the occlusion of the teeth depends on a number of conditions. I shall discuss two influences that are important from a clinical viewpoint.

Read at the Eightieth Annual Midwinter Meeting of the Chicago Dental Society, February 22, 1944.

1. Muscular pressure. There is no disputing the fact that pressure of the tongue, lips and cheeks has much to do with the size, form and relationship of the dental arches. When the mouth is closed, the tongue fills the lingual portion of the oral cavity. Clarence T. Simon, director of the speech clinic of Northwestern University, recently stated that "in one minute of conversation at average speed, the tongue is involved in eleven hundred to twelve hundred movements." Practically every movement of the tongue exerts pressure on the lingual surfaces of the mandibular teeth and, to some extent, upon the maxillary teeth, as well as upon the hard palate. This tongue pressure stimulates development of the mandibular dental arch and, principally through occlusal contact, also influences the size and shape of the maxillary arch. Normally, the tongue pressure is balanced by the restraining influence of the lips and cheeks in contact with the labial and buccal surfaces of the maxillary teeth. The teeth erupt and their investing tissues develop between these two muscular forces, the tongue on the inside and the lips and cheeks on the outside of the dental arches. Unless these forces are properly balanced, there will be abnormal development of the dental arches.

There are a number of factors which may interfere with a normal balance of muscular pressure on the dental apparatus. Among these are: excessive pressure in a particular area or direction by the tongue; abnormal respiration; sucking habits involving the tongue, cheeks, lip,

thumb or fingers, pacifier, blanket, etc.; posture habits, including sleeping postures, and imitation of persons having marked facial deformities.

2. Function of the teeth. The deciduous dentition develops rapidly and serves as a chewing mechanism during the early years of life when rapid growth is taking place. During this period, vigorous use of these teeth in normal function is essential to the normal lateral growth of the dental arches and for the normal vertical development of the face. Normal occlusion and function of the deciduous teeth make possible a normal occlusion of the permanent teeth and normal facial development. Malocclusion of the deciduous teeth presages malocclusion of the permanent dentition. Figures 1 and 2 demonstrate the logic of this statement.

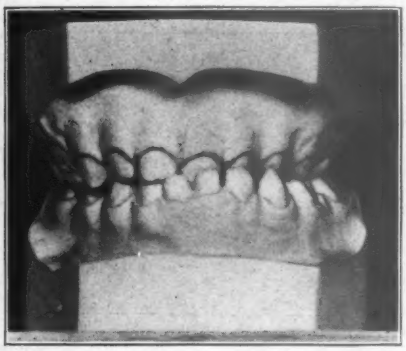

Fig. 1.—Malocclusion of deciduous teeth, presaging malocclusion of succeeding permanent teeth.

If the truth of the foregoing statements is realized, the correction of malocclusion of the deciduous teeth must be recognized as an important responsibility. The best time for orthodontic movement of deciduous teeth is when their roots are fully developed and there is a minimum of development of their permanent successors.

Included in the requirements for the development of normal occlusion are teeth of normal size and normal proximal contacts. Many deciduous teeth require filling. The fillings should restore the normal size and contour of the teeth, and normal proximal contact should be provided. The dentist who does not accept and fulfil his professional obligations in this important matter should realize that, in all probability, he is contributing to an unsatisfactory condition of the patient's chewing mechanism and to malocclusion of the permanent teeth as well.

Proper care of the deciduous teeth, both at the dental office and at home, is important in the development of occlusion. Few will, I believe, object to the statement that deciduous teeth are universally neglected and mistreated. Perhaps the operator does not like to deal with the dental problems of young children, whose behavior in the operating chair may be difficult; or perhaps he feels that the work is unprofitable from an economic standpoint. In many cases, the child's parents do not understand the importance or appreciate the value of good dental care for "baby teeth," and perhaps the dentist does not feel

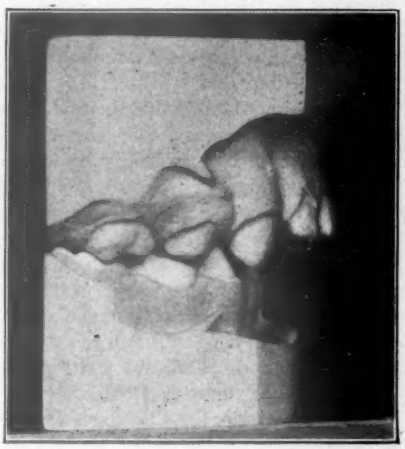

Fig. 2.—Malocclusion of deciduous teeth. Unless corrected, this same type of malocclusion may be expected in the permanent dentition.

inclined to correct such erroneous ideas. Whatever the reasons, we know that too many deciduous teeth are neglected, too many are lost prematurely without an attempt to maintain correct arch form, and too many are kept in the dental arches too long. We see too many fillings which do not meet the requirements laid down by G. V. Black. We see single amalgam fillings occupying approximating cavities in two deciduous molars. Why any dentist should join two deciduous molars with one filling is beyond understanding. He should know that these teeth should not be immobilized and that gingivitis of the interproximal tissues will be a certainty, and he should remember that one of these teeth normally is shed a whole year in advance of the other.

Dentists should prepare themselves to deal with their young patients and to give them their best professional services. Lack of a genuine desire to treat children and carelessness in operative procedures are responsible for many cases of malocclusion of the teeth which lead to lifelong handicaps.

The following routine for the examination and record of children's teeth is recommended. A suitable card should be used. The dentist should not rely upon his memory in this matter. The card should provide space for the following data:

Date
Name of patient
Name of parents
Address
Telephone number
School (grammar, high, private, etc.)—school hours
Age of patient (in years and months)

The card should carry a chart of the teeth and all carious areas should be indicated.

The following items should be given consideration:

1. The general arrangement of the teeth and form of each dental arch should be noted.

2. The teeth should be counted; for, in some cases, there is an anomaly in number.

3. The patient should be asked to close the teeth so that the occlusion may be examined.

4. The mesiodistal relationship of the dental arches should be noted and recorded, each side of the mouth being considered separately.

5. Cross bite, open-bite areas, excessive overbite and abnormal conditions of the soft tissues should be noted.

Inquiries should be made regarding injurious habits which may be factors in malocclusion. If such habits are evident, prompt action should be taken for their correction.

Fig. 3.—Residual fragments of deciduous roots, which may cause malocclusion or become sources of infection.

In addition to the above-mentioned items, full-mouth roentgenograms should be made as soon as is convenient. The films should be examined particularly for the following information:

1. The amount of development or resorption of the deciduous roots.

2. The positions of permanent teeth and the degree of development.

3. The presence of supernumerary teeth.

4. Teeth missing.

5. Fragments of deciduous roots present. Such fragments sometimes interfere

with the normal development of occlusion, and they may become sources of infection. (Fig. 3.)

A careful study of the films may indicate the advisability of subsequent radiodontic examination. Usually, these should be made at intervals of six months.

It is well to take impressions of the teeth and make casts at intervals of six months in order to detect incipient abnormal development. Plaster casts, to be of real value, must be made from accurate impressions. I have examined hundreds of casts submitted to me for examination and advice and only a few of them have been accurate reproductions of the teeth or of the occlusion. In nearly every case, there was a statement that "I merely took snap impressions," which evidently was the excuse of a careless or incapable operator. Such casts are useless for study or as records.

The genuine interest of the operator who keeps accurate and adequate records of his young patients will be a most important factor in preventing malocclusion of the teeth.

The care of deciduous teeth by the patient or his mother is an important matter, and the dentist should offer advice and instruction in oral hygiene at every opportunity. The following health measures are included in an exhibit prepared by the Bureau of Public Relations, American Dental Association, on display at the Smithsonian Institution in Washington, D. C.[1]

1. Frequent and thorough inspection and care of the teeth and mouth by a dentist, beginning when the child is 2½ or 3 years of age and continuing throughout life.

2. A wholesome and well-balanced diet throughout life, especially during the early formative years.

3. Thorough cleansing of the mouth during each meal by eating fibrous, cleansing foods and by correct toothbrushing after each meal.

This outline of dental hygiene is fundamental. Statement 1, which refers to "frequent and thorough inspection and care of the teeth," implies that the dentist will thoroughly inspect and properly care for the teeth. Statements 2 and 3 contain wholesome advice, and I am pleased to note that an appeal is made for "correct toothbrushing after each meal." For many years, I have strongly advocated this plan, but, after discussing the subject with patients and their parents on many occasions, I am convinced that the average person believes that he is taking adequate care of his teeth if he brushes them before breakfast and again just before retiring at night.

Let us consider the result of this routine. A person brushes his teeth in the morning and probably within a few minutes he is at breakfast. Then he rushes to school or to work and all morning there are particles of food lodged between the teeth and in the occlusal grooves. This material decomposes and forms an ideal medium for the process of dissolution of tooth enamel. At noon, another meal is eaten and more food particles are added to those in the mouth. Then there is the evening meal, which leaves another supply of food

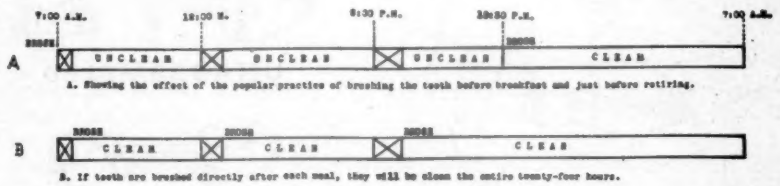

Fig. 4.—Plans for toothbrushing.

particles in positions vulnerable to caries of the enamel. Finally, at bedtime, which we shall estimate as 10:30 o'clock, he brushes his teeth. The diagram in Figure 4, covering a twenty-four hour period, illustrates the situation.

Regardless of the numerous theories and ideas which have been presented on the subject, caries always occurs in what G. V. Black termed "unclean areas." We should do all that we can to encourage our patients in the matter of oral hygiene, and this should include "correct toothbrushing after each meal."

Incipient malocclusion is sometimes the result of the loss of the normal mesiodistal width of deciduous teeth, particularly molars, due to caries. In many of these cases, sufficient separation, followed by fillings of correct size and shape, will correct the condition.

When deciduous teeth are lost prematurely, the teeth distally from the position formerly occupied by the lost tooth move mesially and the teeth mesially from this area sometimes move distally. At any rate, they will not continue in normal forward development, because of lack of proximal contact. When this occurs, there is a condition of malocclusion and a lack of harmony between the developing dental arches. A space maintainer might prevent a greater degree of malocclusion in this particular area, but the best treatment would be to correct the malocclusion by restoring the space and then applying a space maintainer of the proper size, instead of maintaining a space which is too small. In cases wherein such treatment is not considered expedient, an appliance may be used to maintain the existing space and prevent further drifting of the teeth. The principle involved, however, is that the space maintained should equal that of the lost tooth (or teeth) and not be merely sufficient for the tooth (or teeth) expected to erupt into this space, particularly in the case of deciduous molars.

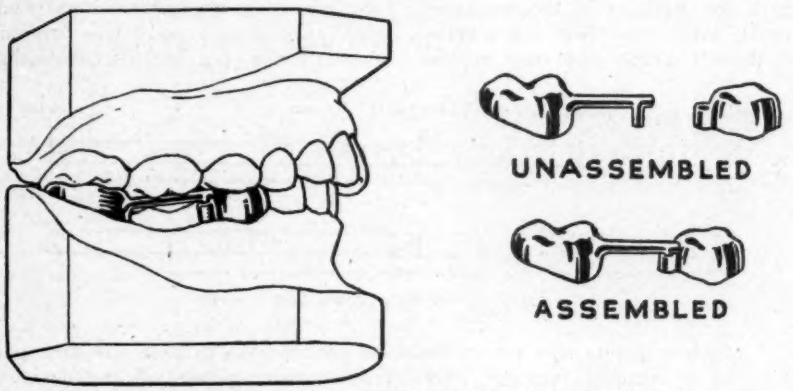

Fig. 5.—Space maintaining appliance. (Courtesy R. C. Willett.)

Space maintainers may be made in various forms. Generally speaking, a space maintainer should have the following qualifications:

1. It should maintain the correct mesiodistal width of the lost tooth (or teeth).

2. It should be attached to or supported by a tooth on each side of the space to be maintained.

3. It should not be a rigidly attached appliance, but should permit movement of the teeth to which it is attached, under stress of mastication.

4. It should give occlusal support to

the teeth of the opposing jaw and afford chewing function if possible.

5. It should not interfere with the eruption of the permanent tooth (or teeth) into the space maintained.

I strongly recommend the type of space maintaining appliances developed by R. C. Willett and described in his papers on "Prevention and Interception of Malocclusion of the Teeth."[2] (Fig. 5.)

The development of the permanent teeth begins early in life, and usually all of the permanent teeth, except the third molars, have erupted and assumed their positions in the dental arches by the age of 12 years. The development of occlusion of the teeth, therefore, occurs principally during the first twelve years of life. This development is coincident with the growth of all tissues associated with the dental apparatus, including the nose, maxillary sinuses, facial bones and muscles. If the development of the occlusion of the teeth is normal, we may confidently expect that the development of the entire lower half of the face will be normal. If malocclusion of the teeth exists, normal development of the associated tissues will be interfered with and the degree of the resulting deformity will be in direct relation to the type and severity of the malocclusion.

In view of the preceding remarks, the following facts must be evident:

The deciduous teeth should receive the same degree of dental service as the permanent teeth. Fillings should restore the normal size and contour of these teeth, particularly the molars.

Unless there is a normal relationship of the maxillary and mandibular dental arches, it will be impossible for the permanent first molars to erupt in their normal occlusal relations. If the permanent first molars are not correctly occluded, a normal arrangement of the other permanent teeth cannot occur.

Malocclusion of the deciduous teeth should receive corrective orthodontic treatment at the age most favorable for obtaining the best results.

The proper care of deciduous teeth will do much to assure better occlusion of the permanent teeth and more normal development of the entire lower half of the face.

After the completion of the deciduous dentition and before the eruption of the permanent teeth, there is a period of about four years. During this time, there is a continued development of the permanent teeth, as well as some absorption of the deciduous incisor roots. There is also a considerable forward as well as lateral development of both dental arches and of all the tissues associated with or influenced by such movement. Space is provided for the first permanent molars, and spaces which may have been present between the deciduous molars and cuspids are eliminated, with resulting proximal contact of these teeth.

There is considerable variability within the normal range of the time of eruption of permanent teeth. Usually, mandibular teeth erupt shortly in advance of corresponding maxillary teeth. One exception to this statement is that, in many cases, the mandibular cuspids erupt two or even three years before the maxillary cuspids appear. I shall use the chronologic order and age of eruption according to the teachings of G. V. Black.

At the age of 6 years, the permanent first molars erupt. In most cases, these teeth erupt directly posteriorly to the deciduous second molars. Occasionally, first molars erupt in an abnormal buccolingual relation. This condition, which is a serious handicap to function and to normal development, should be corrected as early as expedient.

Some claims are made that when permanent first molars erupt, their occlusal relation is an end-to-end contact of the cusps, and that this relation is maintained until the deciduous molars are replaced by the bicuspids. It is true that, in many cases, these teeth do erupt

in a cusp-to-cusp occlusion, but my observations lead me to believe that it is usually a matter of only a few weeks before the molars settle into either a normal or an abnormal mesiodistal relationship. I have seen many cases wherein the permanent first molars erupted in a normal relationship, with the mesiobuccal cusp of the maxillary molar fitting into the buccal groove of the mandibular molar. This may have occurred as a result of greater forward development of the mandibular than of the maxillary arch prior to eruption.

At this time, spaces usually are present between the anterior teeth of both arches, for the jaws have been growing in width to provide room for the larger permanent teeth. The central incisors erupt at 7 and the lateral incisors at 8 years of age. The mandibular incisors erupt first and slightly lingually from the line of the arch. Normal tongue pressure moves them to their correct positions. The maxillary incisors erupt labially from the mandibular incisors and are guided into proper contact with those teeth by normal pressure of the upper lip.

If mandibular deciduous incisors are lost prematurely, the dental arch will not grow sufficiently in width to provide space for the permanent incisors and there will be crowding of these teeth. This condition usually induces a narrowing of the maxillary arch, with crowding of the teeth and probably excessive overbite. Premature loss of the maxillary deciduous incisors is not such a serious matter because the maxillary teeth overhang the mandibular teeth whose positions and function promote and influence the growth of the maxillae.

There is now a period of two years without any change in the dentition. During this time, there is further development of all the permanent teeth and resorption of some of the deciduous roots. There is also a continued forward development of the dental arches coincidental with the development of the second molars.

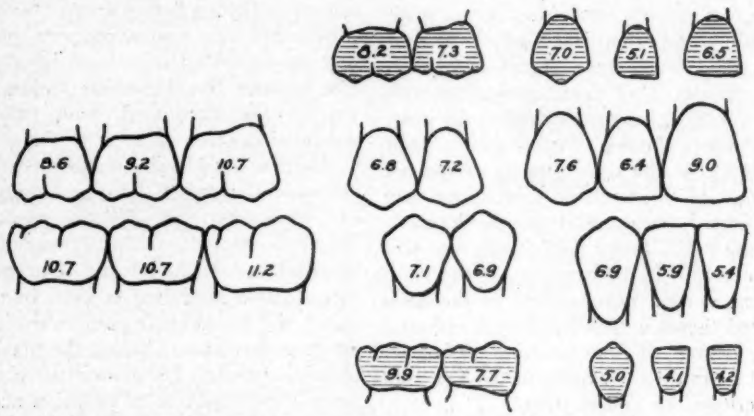

Fig. 6.—Diagram showing (in millimeters) average mesiodistal widths of deciduous and permanent teeth.

The exchange of deciduous molars for bicuspids, an important phase of development, is a much more complicated matter than the exchanges in the incisor region; for the permanent incisors erupt into an area formerly occupied only by deciduous teeth. The jaws have been increasing in width for some time previous to their eruption, and they are

guided to position by the muscular pressure of the tongue and lips, between the deciduous cuspids.

However, the bicuspids erupt between two areas occupied by the permanent teeth and considerable bony tissue. At the age of 10 years, when the first bicuspids are due to erupt, there are well-developed permanent molar regions toward the distal aspect and well developed anterior regions occupied by central incisors which have been in place for three years, lateral incisors which have been in place for two years, deciduous cuspids and developing permanent cuspids. When we consider the normal forward development in the permanent molar region, we

Fig. 7.—Normal arrangement of deciduous molars and cuspids.

Fig. 8.—Loss of mandibular deciduous first molar. At this time, the corresponding maxillary deciduous molar should be extracted.

must realize that it was a wise provision on the part of Nature that the bicuspids should be smaller than the deciduous molars or probably they would not have sufficient eruptive force to assume their places in occlusion.

Normally, the mandibular deciduous first molar is lost at 10 years of age and the first bicuspid erupts. Then, the maxillary deciduous first molar is shed and the first bicuspid erupts. A year later, the same procedure occurs in the case of the second bicuspids.

A maxillary deciduous first molar must be lost from the arch at approximately the time that the corresponding mandibular first molar is lost or malocclusion will result. Normal development will not occur if there are deciduous molars in one jaw and bicuspids in the opposing jaw (on the same side of the mouth), for the teeth will not be harmonious in size. It is a mistake to believe that all deciduous molars should be kept in the arches as long as possible. There is a definite time when each deciduous molar should be lost.

The following is quoted from a published report[8] of a clinic which I presented at the 1916 annual meeting of the Illinois State Dental Society:

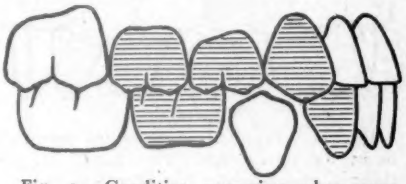

Fig. 9.—Condition occurring when maxillary decidous first molar is allowed to remain in arch too long.

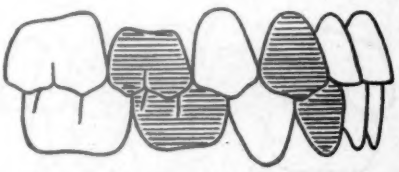

Fig. 10.—Case in which maxillary molar was extracted at correct time (as soon as mandibular first bicuspid was fully erupted) and development is normal.

Rule: As soon as a lower bicuspid fully erupts, the corresponding deciduous upper molar should be removed from the arch.

The time for the extraction of a deciduous upper molar should not be determined by the physical condition of the individual tooth, but by considering the relation of that tooth to the development of the jaws. A deciduous upper molar, although perfect in structure, may help to produce malocclusion, if allowed to remain too long in the arch. It is important to keep the lower deciduous molars in the arch until the bicuspids are

due to erupt, and just as important to remove the deciduous upper molars at the right time.

Figure 6 shows the average sizes, in millimeters, of the deciduous and permanent teeth.

The accompanying figures illustrate the exchange of deciduous molars for bicuspids. The permanent molars are shown in normal occlusion. Actually, in some cases, the molars might be in malocclusion, but, in any event, the principle that I wish to present remains the same. These drawings are made to scale of average teeth and the deciduous teeth are lined horizontally.

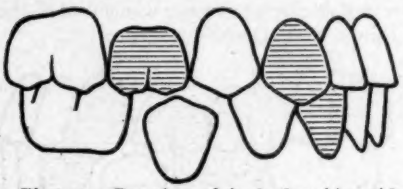

Fig. 11.—Eruption of both first bicuspids in normal relationship. The mandibular deciduous second molar has been lost and the second bicuspid has erupted. At this time, the corresponding maxillary deciduous molar should be extracted.

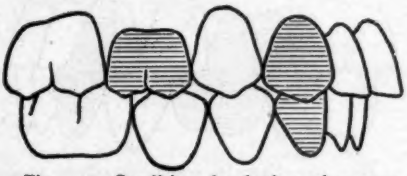

Fig. 12.—Condition developing when maxillary deciduous second molar is allowed to remain in arch too long.

Figure 7 shows all of the deciduous molars in normal occlusion.

In Figure 8, the mandibular deciduous first molar has been shed and the first bicuspid has erupted. At this time, the maxillary deciduous first molar should be removed.

Figure 9 indicates what will happen if the maxillary deciduous first molar is allowed to remain in the arch. The natural forward movement of the posterior teeth closes up the spaces in the mandibular arch and the maxillary anterior teeth are held labially from their normal relation with the mandibular teeth.

Figure 10 shows the result if the maxillary deciduous first molar is extracted at the proper time. The normal relation of the anterior teeth has not been disturbed and there is harmony of the dental arches.

In Figure 11, the mandibular deciduous second molar has been lost and the second bicuspid has erupted. At this time, the maxillary deciduous second molar should be removed.

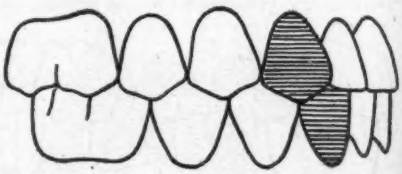

Fig. 13.—Case in which all deciduous molars were shed at correct times and development is normal.

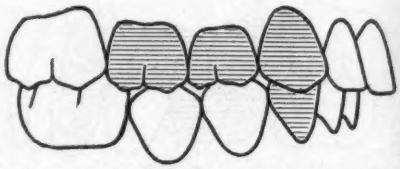

Fig. 14.—Case in which both mandibular bicuspids have erupted and both maxillary deciduous molars are still in arch. This is the worst possible combination of these teeth.

Figure 12 shows what occurs if the maxillary deciduous second molar is left in the arch. If the permanent molars are in normal occlusion, the maxillary anterior teeth are held too far labially to occlude normally with the mandibular teeth.

Figure 13 illustrates a normal arrangement of the teeth following a normal exchange of deciduous molars for bicuspids. The anterior teeth have maintained their normal relations and the molars have moved forward to offset the discrepancy

in the space formerly occupied by the deciduous molars, producing proximal contact of the bicuspids.

Figure 14 shows both maxillary deciduous molars and both mandibular bicuspids in place. This is the worst possible combination of these teeth. The maxillary deciduous molars occupy too much space in the arch, with resultant labioversion of the maxillary anterior teeth. Under these conditions, the posterior teeth receive much more than their normal share of occlusal stress, and therefore there is a lack of normal vertical development in the posterior segments of both arches. At the same time, the anterior teeth, being out of occlusion, become extruded and a deep overbite may result.

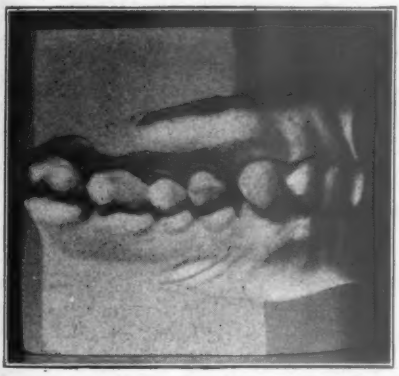

Fig. 15.—Malocclusion possible result of prolonged retention of maxillary deciduous molars.

Figure 15 illustrates a malocclusion which may have been caused by the prolonged retention of maxillary deciduous molars.

Some orthodontists who realized that the mesiodistal width of a deciduous molar was preventing the development of normal occlusion, and who did not feel warranted in extracting a sound tooth that was useful in mastication, have compromised by grinding away the marginal ridges of the deciduous molar, as shown in Figure 16.

I cannot subscribe to this procedure. The tooth will be sensitive to thermal changes, food will lodge between the teeth and there is a possibility of caries or even death of the pulp. If the pulp dies and an abscess results, the deciduous roots will not be resorbed. In any event, the wide spreading roots of the deciduous molar remain and will keep the approximating teeth apart, and this may cause an abnormal inclination of those teeth. Furthermore, the retention of this deciduous molar interferes with the eruption of the succeeding bicuspid, which is needed in the development of occlusion.

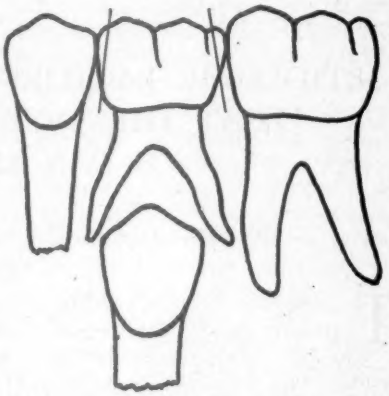

Fig. 16.—Reduction of size of deciduous molars by grinding. This procedure, practiced by certain operators, is not approved.

At the age of 12 years, the second molars and the cuspids erupt. The forward growth of the dental arches has provided room for the second molars, and it seems probable that both arches have also grown to make room for the cuspids, which are larger than the deciduous cuspids. Some writers have stated that the cuspids erupt into part of the space formerly occupied by the deciduous first molars, but this seems improbable and illogical when we recall that the first bicuspids normally erupt two years earlier.

The premature loss of deciduous cuspids is responsible for many cases of malocclusion. Usually, the posterior teeth move forward, and sometimes the entire cuspid space is eliminated. As soon as a deciduous cuspid is lost prematurely, steps should be taken to maintain the normal arch form. If deciduous cuspids are retained in the arches too long, the permanent cuspids are delayed in eruption and sometimes they are forced to erupt in malocclusion.

Third molars are due to erupt from the age of 17 to 25 years. Their influence on the occlusion of the teeth is debatable and I shall not attempt to discuss that problem at this time.

It has been stated by authorities that 75 per cent of existing malocclusion of the teeth might have been prevented. Malocclusion can be lessened a great deal if the deciduous teeth receive adequate care and treatment, if injurious influences are eliminated and if there is a normal exchange of deciduous for permanent teeth.

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ETIOLOGIC FACTORS IN PERIODONTOCLASIA THAT THE AVERAGE PRACTITIONER CAN ELIMINATE

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IN presenting the facts concerning the causative factors in periodontoclasia to the average practitioner, it is important that these facts be set down in a clear, simple manner. The general practitioner is not interested in detailed descriptions of a highly scientific nature, but rather is desirous of obtaining definite knowledge that can be employed in his daily practice. Neglect of some elementary considerations of periodontology is probably due to misconception or lack of knowledge, this often resulting from too many profoundly academic presentations.

Oddly enough, periodontology is the simplest, yet most essential of the branches of dental science and the one that promises the favorable results of logical therapy. The therapy employed must, however, be based on a clear-cut understanding of the etiology of periodontoclasia.

It is the purpose of this paper to present some of the important local etiologic factors in periodontoclasia with a view to interesting the average general practitioner in a better service to his patients.

Most important among these "local" factors is oral sepsis, "poisoning of the tissues of the mouth by the absorption of putrefactive products." Clean, well-cared-for-mouths are not highly susceptible to any form of periodontal disease, as high tissue resistance is an accompaniment of oral cleanliness and lessens the possibility of gingival infection from the constant presence of many pathogenic

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oral bacteria. Simple, elemental and necessary as oral hygiene is, the prevalence of unhygienic mouths is a definite indication that many people are not cognizant of oral uncleanliness as an etiologic agent of periodontoclasia. The reason for this is probably that the efforts of the dental profession are not so effective as they should be in promoting the program of oral health and early periodic dental care. The average dental practitioner could do a great deal to remedy this situation if he were to insist on giving a prophylaxis, with mouth health instruction, to every patient that comes into his office and to emphasize, possibly exaggerate, the results of oral neglect.

Calculus, materia alba and mucin plaques are the next most important source of irritation. Calculus is recognized as of two types—salivary and serumal. The former develops above the gingival margin, consisting of mineral salts embedded in a bacterial and fungus matrix attached to the teeth, and causing inflammation of the gingivae. This matrix below the gingival margin is of similar composition and is the result of a preceding inflammation. It is formed by calcification of the inflammatory exudate about the necks of the teeth. Materia alba is a soft white deposit composed of food débris, mucus, dead epithelial cells, purulent matter and various types of bacteria. It forms around the necks of teeth and its presence usually results in a gingivitis. Mucin plaques are found in protected or uncleanly areas of the teeth. These plaques of mucin precipitated from saliva form an ideal medium for the growth of oral bacteria in general and *Lactobacillus acidophilus* in particular. The eventual havoc wrought by the presence of these deposits when left undisturbed for long periods is not fully impressed on the minds of practitioners and, as a result, their removal is a secondary factor in the care of patients. The prompt and uneventful recovery of tissues relieved of this irritation should be ample incentive to practicing dentists to thoroughly clean all mouths.

The next factor, faulty dental operations, constitutes one of the major irritational factors in gingival inflammation and osteoclastic resorption. It is in the fields of crowns and bridgework, partial denture prosthesis and operative dentistry that the greatest amount of damage is done. Too little attention is paid to finishing the gingival margins of crowns and abutment inlays, with the result that small imperfections remain to act as constant sources of irritation to the sensitive gingival tissues, in addition to being nidi for the accumulation of oral débris and bacteria, with resultant added irritation and recurrence of decay. These irritational factors are many and varied; and if the average practitioner recognized their importance, he would be much less inclined to place crowns and bridgework which are obviously deficient in technical workmanship and to which the tissue reaction is unfavorable.

In partial denture prosthesis, strict attention should always be given to the stresses and strains to which clasped teeth are subjected. The large percentage of clasped teeth that become loosened after the wearing of partial dentures, ultimately requiring either discontinuance of the use of the appliance or removal of the involved teeth, indicates the need for more scientific knowledge about these necessary prostheses. The daily removal and cleaning of these appliances is another point that the dentist should stress. Too often, the dentist sees appliances that are seldom taken out of the mouth and cleaned. The patient usually states that the dentist does not give instruction in the daily cleaning of dentures.

Poor operative dental work is probably accountable for the majority of cases of localized periodontal involvement. The danger from crowns and inlays with

gingival excess has already been mentioned. Similar undesirable conditions are produced by gingival overextension of amalgam, porcelain, gold or cement fillings.

In addition to factors just mentioned, there is the question of contact point of proximal fillings, a subject constantly referred to and written about, but too often neglected. The point form and tightness of the contact point are important factors in the elimination of interproximal gingivitis. Injury or systemic disease may be conducive to weak contacts, which allow for the lodgement and retention of food, with later an inflammatory reaction. As these contacts become weaker, an open contact results eventually. With open contacts, the irritation becomes proportionally greater and the damage more nearly reaches the stage of irreparability.

It has been suggested that these conditions are insignificant and it is illogical to think that these irregularities actually cause periodontal disease. Cases have been shown wherein these conditions were present, but disease did not result. It is well to remember that the time element enters into the picture, and that, over a period of time, a small but constant irritational process will have undesirable effects. The fact that an inherent factor in certain individuals prevents the production of disease indicates a tendency of the involved tissues to remain in a state of health in spite of irritational conditions.

These arguments are merely attempts to justify certain technical deficiencies. Practicing dentists will have to do better, because the increased dental education of the public is making for a knowledge of where the fault lies. In addition, the correction of these few specific etiologic factors lies within the jurisdiction of the dentist himself and involves more consideration of some of the basic principles taught in dental schools.

SUMMARY

As yet, there has not been found any one specific etiologic factor in periodontoclasia, even though several contributing factors have been demonstrated. Various investigators have attributed more importance to some of these factors than to others. There are some rather obvious local factors that for many reasons have not been fully grasped by many members of the dental profession: (1) oral sepsis; (2) deposits on the teeth, and (3) faulty dental operations: (a) operative dental work, (b) crowns and bridgework and (c) partial denture prosthesis. These are conditions which the dentist can and should correct promptly. It is suggested that dentists pay more attention to these conditions as the public is becoming dentally educated and is beginning to realize that the retention of teeth is dependent on the health of the supporting tissues.

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EFFECTS OF BUFFERED LETHAL DOSES OF FLUORIDE ON GUINEA-PIGS

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VOLUMINOUS reports have been written on the toxic effects of excessive fluoride intake, which is responsible for chronic endemic fluorosis. Recently, such investigators as Harootian[2] and Branson[3] have pointed out the need of a small daily intake of fluoride in food, water or bone meal to insure greater immunity to caries in growing or fully erupted teeth, while Bibby[1, 6] advocates topical application.

Few if any of the systemic effects, other than on the teeth and bones, have been studied in human beings receiving minute daily doses.

Sollmann[4] states that the fatal dose of sodium fluoride for mammals is 0.5 gm. per kilogram of body weight, when taken orally. To test the effects of fatal doses of several fluoride salts, a series (twenty) of experiments were conducted on guinea-pigs. The procedure was as follows:

The guinea-pigs were weighed and their normal activities noted. No pigs were selected that gave any indication of abnormality.

The dosage given the first series of guinea-pigs was double the lethal dose; that is, a 500-gm. pig was given not 0.25 gm. of sodium fluoride, but 0.50 gm., double the fatal dose as indicated by Sollmann.[4] This was done to insure a positive lethal dose, as a very small amount was lost during administration. The fluoride was given orally in one-fourth ounce of water by placing a syringe in the animal's pharynx and injecting the fluid, forcing the pig to swallow. The results are tabulated on the accompanying chart. Two animals were allowed to die to establish the potency of the drug, and these served as controls.

The next step was to administer a buffering agent with fluoride which would inactivate the drug in the gastrointestinal tract. Since calcium and magnesium salts will remove fluoride from drinking water, these agents were used. Each guinea-pig was given a double lethal dose of sodium fluoride, according to the individual weight of the animal, plus an equivalent amount of calcium carbonate and one-half an equivalent amount of magnesium oxide. The reactions to this dosage were very gratifying.

Ten minutes after administration, the guinea-pigs were active and evidently normal. Within two hours, most of the animals were eating normally, an evidence of well being. Persons at this institution who are well versed in the habits of these animals state that lack of interest in food is a certain sign of sickness or malaise.

In several instances, the guinea-pigs developed a respiratory wheeze, which persisted for several hours. This was attributed to aspiration of the administered mixture into the air passages. A few animals exhibited reduced movement for some time after handling.

A second series of twelve animals was given similar treatment, except that the magnesium oxide was left out, only a

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EXPERIMENTAL DATA

No.	Weight of Animal Gm.	Fatal Dose Gm.	Administered Dose Gm.	Sex	Color	Effects After Administration: 10 Minutes	20 Minutes	30 Minutes	40 Minutes	1 Hour	2 Hours	1 Day*
						Group 1						
1	630	0.315 NaF	0.63 NaF	♂	Brown	Decreased activity	No movement	Convulsions and coma	Death			
2	485	0.243 KF	0.485 KF	♂	White	Normal	Decreased activity	Lassitude	Coma	Death		
3	600	0.30 NaF	0.30 NaF 0.60 $CaCo_3$ 0.30 MgO	♀	Brown	Normal	Quiet wheezy	Normal movement	Normal	Eating, breathing	Normal	Normal
4	620	0.31 NaF	0.62 NaF 0.62 $CaCo_3$ 0.30 MgO	♀	White	Normal	Normal	Normal	Normal	Eating	Eating	Normal
5	562	0.28 KF	0.56 KF 0.56 $CaCo_3$ 0.23 MgO	♀	Albino	Quiet	Normal	Normal	Active	Eating	Normal	Normal
6	480	0.24 NaF	0.48 NaF 0.48 $CaCo_3$ 0.24 MgO	♀	Brown	Quiet	Same	Same	Same	Unusually quiet	Same	Inactive but eating
7	650	0.32 NaF	0.65 NaF 0.65 $CaCo_3$ 0.32 MgO	♀	Black	Normal	Same	Same	Quiet	Eating	Normal	Normal
8	500	0.25 KF	0.50 KF 0.50 $CaCo_3$ 0.25 MgO.	♂	White	Very quiet	Quiet	Quiet	No activity	Quiet	Moves about	Eating
						Group 2						
9	460	0.23 NaF	0.46 NaF 0.90 $CaCo_3$	♂	Brown	Active	Active	Quiet	Noisy active	Eating	Normal	Same
10	525	0.26 KF	0.525 KF 1.0 $CaCo_3$	♀	White	Quiet	Same	Same	No movement	Quiet	Normal	Eating
11	600	0.30 NaF	0.60 NaF 1.2 $CaCo_3$	♀	Brown	Normal	Noisy	Active	Eating	Normal	Same	Same

11	600	0.30 NaF	0.60 NaF 1.2 $CaCo_3$	♀	Brown	Normal	Noisy	Active	Somewhat quiet	Quiet Eating	Normal Eating	Same	Same
12	480	0.24 KF	0.48 KF 1.0 $CaCo_3$	♀	Black	Normal	Normal	Somewhat quiet	Quiet	Eating	Normal	Normal	Normal
13	470	0.23 KF	0.46 KF 0.90 $CaCo_3$	♂	White	Quiet	Quiet	No movement	Active	Active	Eating	Normal	
14	620	0.31 KF	0.62 KF 1.2 $CaCo_3$	♀	White	Quiet	Slightly active	Moves about	Eating	Normal	Normal	Normal	
15	600	0.30 NaF	0.60 NaF 1.2 $CaCo_3$	♂	Striped brown	Normal	Normal	Normal	Normal	Active	Eating	Normal	
16	560	0.28 KF	0.56KF 1.2 $CaCo_3$	♀	Black	Active	Normal	Eating	Restless	Normal	Normal	Normal	
17	620	0.31 NaF	0.62 KF 1.24 $CaCo_3$	♂	Brown	Quiet	Quiet	Moves about	Active	Eating	Quiet	Normal	
18	480	0.24 NaF	0.48 NaF 1.0 $CaCo_3$	♀	Brown	Seemed normal	Quiet	Noisy	Normal	Eating	Normal	Normal	
19	500	0.25 KF	0.50 KF 1.0 $CaCo_3$	♀	Albino	Normal	Normal	Eating	Resting	Quiet	Normal	Normal	
20	460	0.23 KF	0.46 KF 0.90 $CaCo_3$	♀	White	Very restless	Active wheezing	Quiet	Eating wheezing	Quiet	Normal	Normal	

*Findings at two and three days unchanged.

double lethal dose of fluoride and double that amount of calcium carbonate being administered. No deaths occurred, and the guinea-pigs seemed quite normal one and a half hours later.

The animals were given the usual diet of oat grains, commercially prepared food pellets, fresh greens, such as cabbage and lettuce leaves, and tap water. The food trays were kept filled at all times. Special locks on the cages prevented entry by other persons. The fluoride-fed animals discharged fecal pellets of the usual size and description, except in greater numbers. The experiment was repeated in its entirety at the end of three days with the same guinea-pigs, using the buffered fluoride mixture. No deaths occurred and no toxic symptoms were manifested. The animals were then observed for three weeks.

The fact that the administration of calcium carbonate and magnesium oxide prevented the fluoride from causing death may be explained by the following reaction, which probably occurred in the animal's stomach:

$$2\ NaF + CaCo_3 + 2\ HCl \rightarrow CaF_2 + 2\ NaCl + H_2O + Co_2.$$

The calcium fluoride is relatively insoluble, its solubility being 0.0016 gm. in 100 gm. of water solute at 18° C.[5] Therefore, absorption in the gastro-intestinal tract was probably nil, and the fluoride was eliminated in the feces.

CONCLUSIONS

1. The guinea-pigs administered a double lethal dose of sodium or potassium fluoride died in forty to sixty minutes.

2. Eighteen guinea-pigs administered double lethal doses of sodium or potassium fluoride buffered with calcium carbonate with or without magnesium oxide did not die. These animals exhibited normal activity and resumed regular feeding within an hour and a half after administration of the fluorides.

3. These favorable results of the use

of the buffered fluorides indicate a drastic reduction of toxicity.

4. These conclusions reflect the promising possibilities of using the fluorides in a dentifrice with a safe outcome even though it perhaps might be ingested in small quantities.

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ORAL ASPECTS OF HIGH ALTITUDE FLIGHTS

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WITH the increasing emphasis that is being placed on aviation, it is believed that these oral observations on high altitude flight may be of interest to the profession.

This paper is based on a series of studies of pilots of a photographic reconnaissance squadron who have been under my care over varying periods. Classified as to time of active flying service, they may be divided into three groups: I. A group of seven with a time of fifteen months. II. A group of five with a time of ten months. III. A group of eight with a time of eight months.

The particular type of missions in which they are involved necessitates flights of extremely high altitude over lengthy periods of time. On the basis of altitude and length of time, it is, I believe, a group unique in the Air Corps. The average flight is approximately 35,000 feet, 40,000 feet being seldom exceeded and 28,000 feet being the minimum. The length of time at these altitudes varies from two to four hours. The rate of climb to the optimum altitude is accomplished at a speed of 1,000 feet per minute.

The approximate temperatures in this theater at 30,000 and 40,000 feet are 30° and 40° below zero, respectively, with the tropopause at 30,000 feet. Throughout this study, a close relationship was maintained with the flight surgeon, and the medical histories were correlated in the preparation of data. This was a vain attempt to connect definite oral and bodily symptoms, particularly when aero-embolism was noted.

In considering the oral symptomatology of high altitude flight, it is apparent that a number of logical conditions might be anticipated. It would be expected that the combination of lowered temperature and pressure would elicit the usual thermal response from a pulp that is already bearing the burden of a large metallic filling. The next question arising is: Would the presence of incipient caries cause any untoward reaction on the part of the tooth when it is subjected to these changes of temperature and pressure? Would advanced caries attended by pulpal reaction or definite pulpal degeneration be explicit in their symptomatology? To observe further, could the nitrogen forced from the blood owing to changes in pressure, and which

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is responsible for aero-embolic sequences, be carried into the pulp chamber of the tooth? Even though this nitrogen has a special predilection for bone marrow, the foregoing assumption could be made. Lastly, I was interested in the observance of the effects, if any, on the teeth and gingival tissues, of breathing of pure oxygen. These questions were paramount in my mind throughout sixteen months' association with and treatment of these pilots.

Primarily, I shall discuss my findings as to the thermal reactions of the pulp. Despite the fact that more than 75 per cent of the pilots had at least four large two-surface restorations, there was only one instance in this entire period in which a tooth exhibited definite pain on temperature change. The involved tooth in this case was a lower left second molar. On physical inspection, it presented a large amalgam restoration embracing the occlusal, distal and facial surfaces. There was definite leakage at the facial aspect of the filling. All the associated subjective symptoms of an active hyperemia were manifest, with increasing intensity of pain on attaining high altitude. Therapy consisted in complete removal of the defective restoration and sedation of the pulp by use of a filling of zinc oxide and eugenol. When all active symptoms had subsided, the temporary filling was removed and amalgam was again used to refill the tooth. No base was employed because of inability to utilize a much needed retention point. All reactions subsided and no recurrences were noted. This tooth was pathologically affected and altitude increased the pain.

From the foregoing, I assumed that the teeth have sufficient adaptability to acclimatize themselves to temperature changes in the period of time required to attain the altitudes where such changes are manifest. The same reasoning led me to ignore the factor of high altitude flight in operative dentistry. There is no need whatsoever for other than a judicious use of a base, this being governed by our knowledge of physiology of the tooth. To me, the progress of the caries and the observance of pulp limitations are here of paramount importance.

My attention was now turned to the relationship that might exist between caries, both incipient and well advanced, and altitude flying. This study was facilitated by the arrival of nine new pilots. It was not possible for me to obtain complete dental examinations of these men until three months and many hours of flying time had elapsed for them. Six of these men were in need of dental treatment, a group which I shall further classify into (1) an incipent class and (2) a "well-advanced" class. In each of these classifications were three men.

The men in the incipient class each possessed one, but not more than two small pit cavities with only slight dentinal involvement. The teeth of none of these men had ever caused them any discomfort throughout their flying careers.

In the "well-advanced" group, each of the three men possessed at least two carious lesions that justified the use of a base in the restoration. One of these pilots, on examination, presented (1) a marked overbite with slight gingival impingement, (2) a horizontal type of periodontoclasia, which necessitated the extraction of both upper third molars, and (3) eight carious teeth, six of which required bases and two surface preparations. Again none of the men examined complained of any symptoms whatsoever from the carious teeth throughout their flying histories.

Thus the assumption that changes of temperature and pressure due to high altitudes are sufficient to cause a carious tooth to become symptomatic was, in my mind, clearly disproved. I do believe that perhaps, in isolated instances, these teeth will react, but such cases are too isolated to enable one to say that this is a definite criterion.

Thirdly, I should like to discuss the probability of what we might term an "aero-embolic" tooth. Aero-embolism itself is a condition peculiar to those subjected to sudden pressure changes. These changes force the body nitrogen from the blood into the tissues or, as is more commonly believed, the bone marrow. Could not some of this nitrogen, overcoming natural anatomic handicaps, be forced into the pulp chamber of the tooth? This would no doubt be singular, and intense, lasting pain might be expected. I shall go no further along this point as I have no actual cases to substantiate it. It is of interest, however, and future problems concerning it may arise. Physiologically, it is possible.

The possibility of expansion of trapped air under inlays causing pain, I am inclined to contest. Even if the operator were careless enough to confine large air spaces in cementation of an inlay, the air would have to lie in direct proximity to the pulp for the expansion to be felt, and any inlay so placed would doubtlessly be productive of other pathologic conditions that would have been noticed.

The use of oxygen seemed to be entirely innocuous and no deleterious effects were observed. The feature of especial interest to me here was the impotency of the molecular oxygen so consistently breathed and, in most instances orally, against the organisms of Vincent's angina. While it is not for me to attempt to discuss the vagaries of this disease or elaborate upon the enigmas of it, it seems that the oxygen treatment, empiric or not, depends on the transformation of nascent oxygen to molecular oxygen. Of four cases seen, in none were any results, beneficial or detrimental, noted. These cases were all cured by chromic acid therapy.

During this thirteen months of work, only three complaints were registered. The first has been previously described as the hyperemic tooth. The second was in the mouth of the same patient, consisting of a sharp diffuse pain, occurring only on descent, lasting approximately five minutes and centralized along the inferior and posterior borders of the left masseteric region. The etiology of this pain could have been either (1) loss of vertical dimensions due to overbite or (2) excessive periodontoclasia involving the upper third molar. Owing to the sudden onset and a short history, the latter was suspected and the tooth was extracted, and all symptoms subsided. The final complaint was centered around a devitalized tooth upon which root-canal therapy had been performed about five years previously. During this period, regular transitory flare-ups were experienced. At these times, the discomfort was always increased by high altitude. On removal of the tooth, a small granuloma was found over the apical foramen.

These experiences seem to point to the fact that high altitude changes tend to accentuate the symptoms of any preexisting pathologic process. There is much work yet to be done, and time should bring to light many oral problems in direct relationship to flight.

SUMMARY

1. At no time did a tooth give any thermal response to changes of temperature caused by both atmospheric conditions and respiration of oxygen.

2. Carious teeth in both incipient and well-advanced stages presented no syndrome or reaction to high altitude changes.

3. The possibility of an aero-embolic tooth is one that should not be ignored. No evidence can yet be gathered to prove or disprove the assumption.

4. The constant use of oxygen over lengthy periods of time did not prove to be detrimental or beneficial to the teeth or the oral tissues themselves.

5. All preexistent pathologic states were definitely accentuated by altitude changes.

A NEW CONCEPT OF THE CARIES PROBLEM AND ITS CLINICAL APPLICATION

PART I

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INTRODUCTION

IT seems that two considerations have prevented a proper approach to the study of dental caries. At the beginning of its histologic study, enamel was considered as nothing more than a calcified material not having any organic structures. Correspondingly, when disintegration of that material was observed, only acid was regarded as a probable acting agent. The profession clung for more than a hundred years to the concept that caries is the result of acid action. It has become evident that this concept is not correct.

The second consideration is that the carious cavity was regarded as representing the carious process, and the early stages of the process have therefore been overlooked. A full understanding of dental caries can be attained only if the early stages are studied by the coordination of microscopic, roentgenographic and clinical findings.

LITERATURE

It has been found[1] that pictures similar to those found in incipient enamel caries can be produced by impregnation of enamel of extracted teeth with ammoniacal silver nitrate. The results of acid action on enamel have no resemblance to caries.[2] It has been shown[3] that in enamel caries, acid action may lag behind the carious process proper. Caries may have started to affect the dentin, whereas the transverse striation of the enamel, produced by acid action, is still far from the dentino-enamel junction. The comparison of x-ray pictures of extracted teeth with ground sections of the same teeth[4] proved that certain stages of early dental caries cannot be detected by x-ray examination. The characteristic feature of caries is a yellow pigmentation which appears long before any necrotic cavity develops. From then on, it is found in all stages of the carious process.[5]

Homogeneously calcified, transparent enamel seems to play an important rôle in arresting the progress of invasion of micro-organisms.[2, 6] In a case in which caries entered a lamella, a layer of transparent enamel was found at which the lamella itself ended and which blocked the progress of the caries (8, Fig. 1). Transparent enamel could be produced by acid action on extracted teeth.[2]

Impregnation of the teeth with sodium fluoride has been recommended for the arrest[8] or prevention[9, 10] of caries and for desensitizing tooth surfaces.[11] If teeth are impregnated with a compound containing silver nitrate, positive acidophilus counts can be changed into negative ones.[12]

By examining enamel by different methods,[13, 14, 15, 16] it has been established that the outer part of the enamel is harder than the inner part. Grenz ray examination of ground sections revealed[17, 18] that, in incipient caries, one

Read before the Wisconsin State Dental Society, Milwaukee, March 20, 1944.

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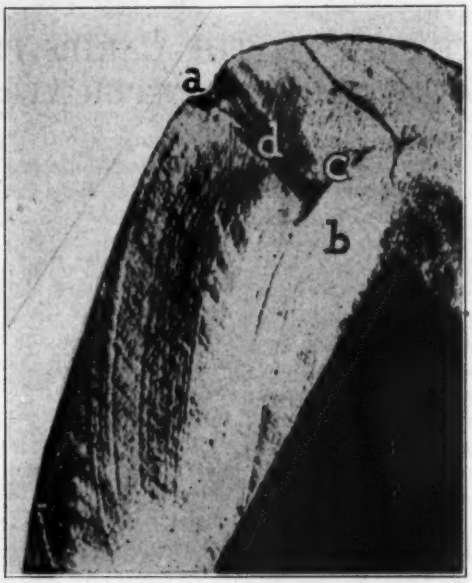

Fig. 1.—a, round discolored depression indicating invaded lamella. A unique finding was the fact that the lamella (d) and caries stopped at a layer of transparent enamel (b). Caries spread horizontally along the incremental line (c), the only organic outlet. (Reproduction from Barker.)

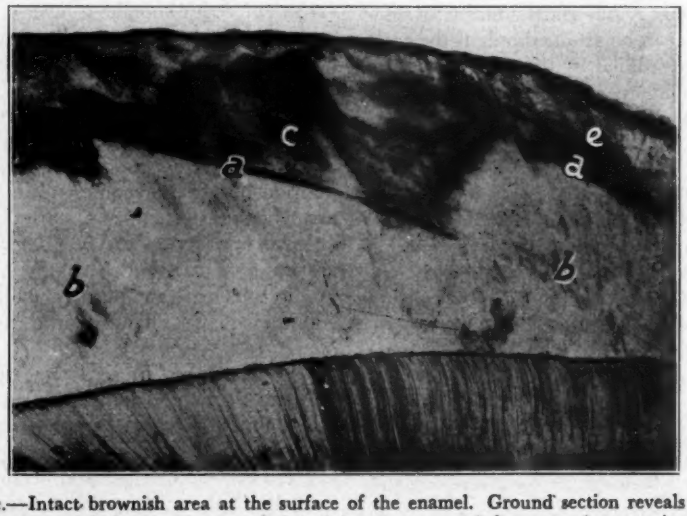

Fig. 2.—Intact brownish area at the surface of the enamel. Ground section reveals that the brownish area and the transverse striation (c) are prevented from crossing two incremental lines (a) where layers of transparent enamel begin (b).

finds decalcification in the deeper parts, while a surface layer of the enamel remains intact. That peculiar condition has been attributed to invasion of the deeper parts by acid-producing micro-organisms.[19]

The circulation of the dental lymph was studied by staining the lymph with solid methylene blue.[20] It was also found, by means of radioactive phosphorus, that traces of lymph circulation are present at the dentino-enamel junction even in pulpless teeth.[21] A dead tract was described leading from beginning caries to the pulp.[22] Acid action on enamel was weakened by adding tricalcium phosphate.[23]

Artificial dental caries has been produced in rat molars by means of food ground to a certain size.[24, 25]

It was suggested and was supported by histologic findings that the prism sheaths cement the calcified parts of the enamel to each other and the tufts cement the enamel to the dentin. The lamellae were supposed to have no physiologic purpose. All these organic structures of the enamel may serve as invasion roads for micro-organisms acting to produce caries.[26]

It was shown that the saliva of a caries-immune dog can sterilize an implanted carious human tooth, while saliva of a caries-susceptible monkey could

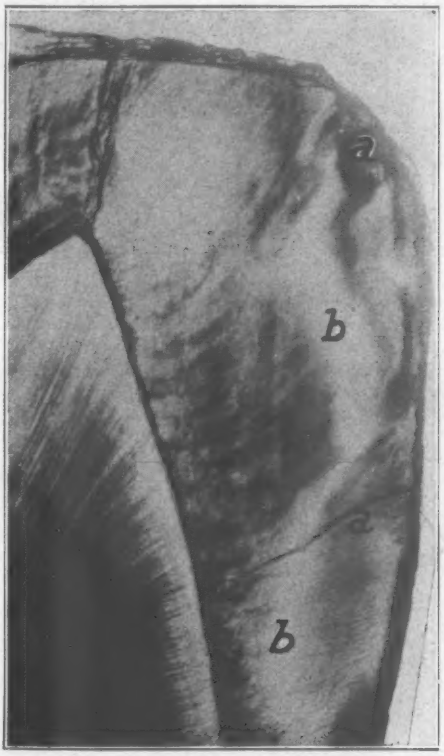

Fig. 3.—Intact brownish area of enamel in a ground section. The brownish area (a) is separated by an irregularly shaped zone of transparent enamel (b) apparently developed in connection with the carious process.

not do it.[27] Atkins[28] found that rinsing with a mouthwash of sodium fluoride brings the acidophilus count down.

FINDINGS

Figure 2 illustrates the ground section of a tooth with an intact surface, but as the spearheading transverse striation (c), stopped spreading deeper at two incremental lines (a), which separated invaded enamel from layers of transparent enamel (b). It shows that acid is present only in places which are reached by the micro-organisms acting with a brownish discolored area at the surface. The ground section revealed that the brownish pigmentation, as well to produce it. Here, all micro-organisms are prevented from spreading into homogeneously calcified enamel. Figure 3 is a

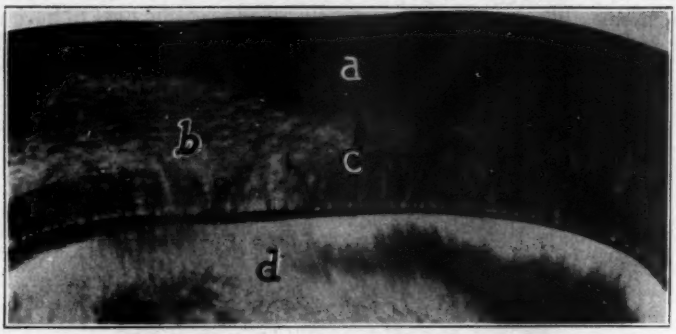

Fig. 4.—Ground section through a brownish area of an intact enamel surface (a). The carious process seems to be arrested, not separated from the intact enamel (b) by a transparent zone. An apparently impermeable lamella (c) ran through the discolored area. d, dentin.

Fig. 5.—High magnification of frontal enamel caries in ground section. a, intact enamel Acid action forming the spearhead produced cloudy enamel and transverse striation (b).

ground section through an intact, but brownish enamel area. The discoloration (a) is limited by an irregular strip of transparent enamel (b). That zone is apparently produced by the action of an acid accompanying the carious process. The areas of transparent enamel in Figures 1 and 2 have been formed during development. They are limited by incremental lines, while the strip in Figure 3 shows irregular boundaries. Figure 4 shows an extended area of brownish enamel (a), leaving a considerable dentinal portion intact (b). The lamella (c) was apparently obstructed; not being passable for the carious process. Neither the dentin (d) nor the tufts could be reached. No zone of transparent enamel was found. This shows apparent arrest of enamel caries without a barrier of transparent enamel. Figure 5 is the reproduction of part of the

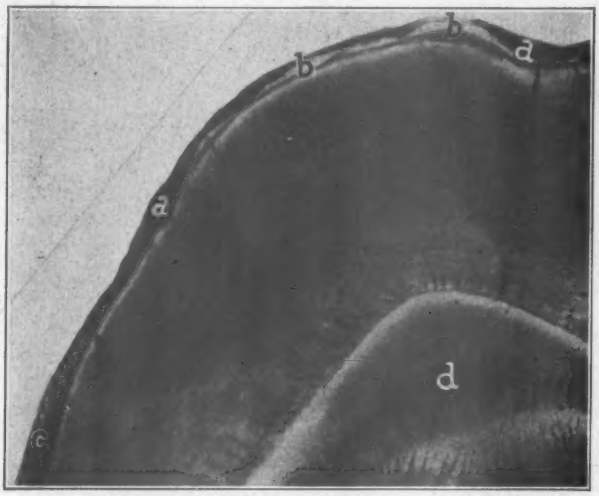

Fig. 6.—Cross ground section along brownish chalky belt near gingival margin. The black areas (a) are brownish. The light strips (b) are transparent enamel. The dark strip (c) is chalky enamel. d, dentin.

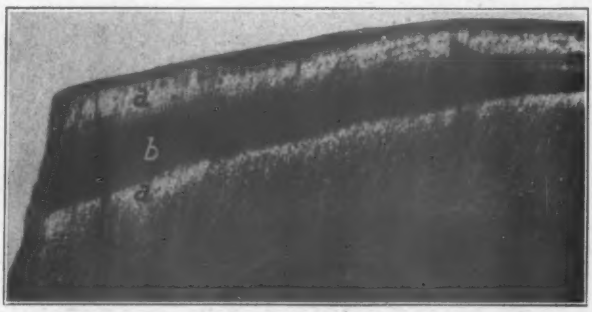

Fig. 7.—Higher magnification of area c, Figure 6. Two lines of transparent enamel (a) are separated by a darker line (b).

enamel of a ground section. The surface was brownish. Cloudy enamel with transverse striation (b) spearheads an area of frontal caries. The transverse striation is more marked than can be

through a molar which showed a brownish strip running around the tooth near the gingival margin. In the black areas, there was brownish discoloration (a). The light areas (b) are transparent

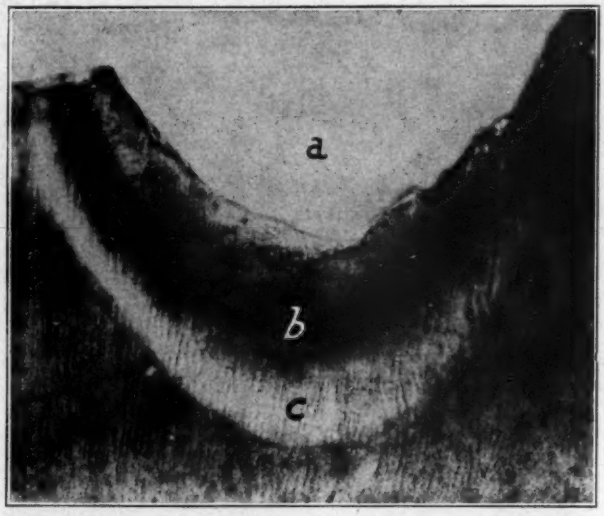

Fig. 8.—Limited area of enamel of extracted tooth exposed to 10 per cent solution of sodium bisulfate for one month. A superficial part of the enamel was dissolved (a). The following layer became dark (b). The following layer became transparent (c).

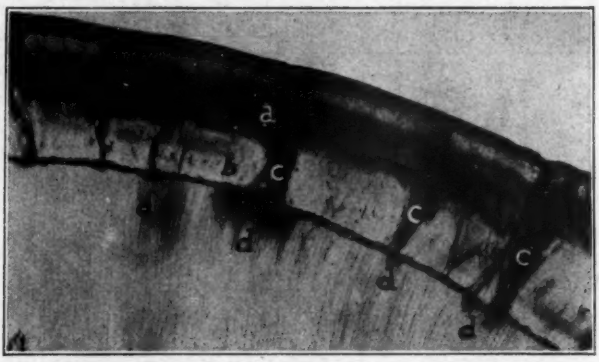

Fig. 9.—Ground section through a tooth with brownish enamel surface. The brownish pigmentation (a) affects the superficial part of the enamel. The dentinal part (b) is intact. A lamella (c) carries the yellow pigmentation into the dentin (d).

produced by acid action on extracted teeth. (Fig. 22, a, intact enamel.)

Figure 6 is a cross ground section

enamel. The dark area (c) was chalky, not brownish enamel. The process is limited to a shallow strip remaining far

from the dentin (d), in spite of the great extent at the tooth surface. Figure 7 is a higher magnification of the chalky part (c) in Figure 6. Two strips of transparent enamel (a) border a strip of opaque enamel (b). The process seems to be characterized by a shift of calcium salts caused by acid action. (Figs. 7 and 8.)

Figure 8 shows how transparent enamel can be produced on extracted teeth by slow acid action. An extracted tooth was covered with sticky wax and a window was left open on the enamel

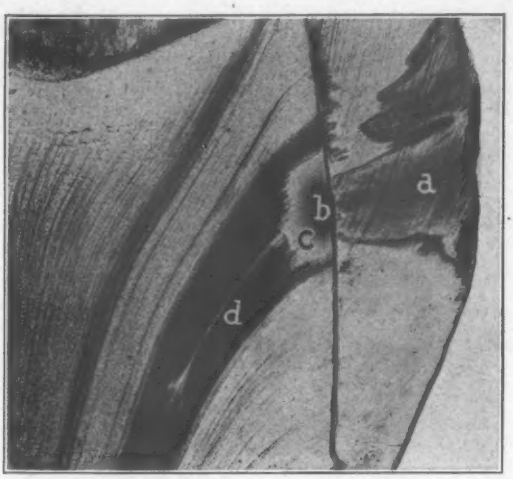

Fig. 10.—Ground section, discolored intact enamel surface. The tip of the frontal enamel caries (a) extends to the dentin. An area of yellow pigmentation (b) is surrounded by transparent dentin (c), which is apparently decalcified. A tract of opaque dentin (d) extends to the pulp canal along the dentinal tubuli.

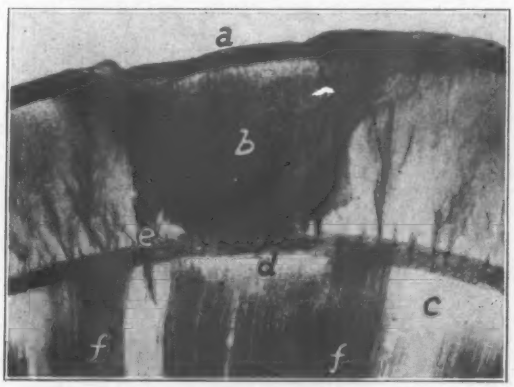

Fig. 11.—Ground section through a brownish tooth area. A depression of the tooth surface at a marks the frontal caries (b). Caries has reached the dentin (c). A minute shrinkage has developed in the dentin (d). Around the lamella (e) and adjoining the shrinkage area, transparent dentin developed surrounded by an opaque area (f).

surface. The tooth was put into a 10 per cent sodium bisulfate solution and left there for one month. The superficial portion of the enamel (a) was dissolved. A deeper strip (b) became opaque. The following strip (c) turned transparent, apparently hypercalcified by the salts penetrating from the opaque strip.

Figure 9 shows a ground section through a tooth with intact brownish enamel. The pigmentation (a) penetrates half way into the enamel. The dentinal part (b) of the enamel is intact, but is cloudy and transversely striated in its peripheral portion. Lamellae (c) run from the surface of the dentin, carrying the yellow pigmentation to the dentin. Around nearly every entrance of a lamella, the dentin is yellow (d). The histologic structure of the dentin is preserved. The tubuli in the affected dentin appear more accentuated than in the intact dentin. This, the carious process proper, does not show up in the x-ray picture.

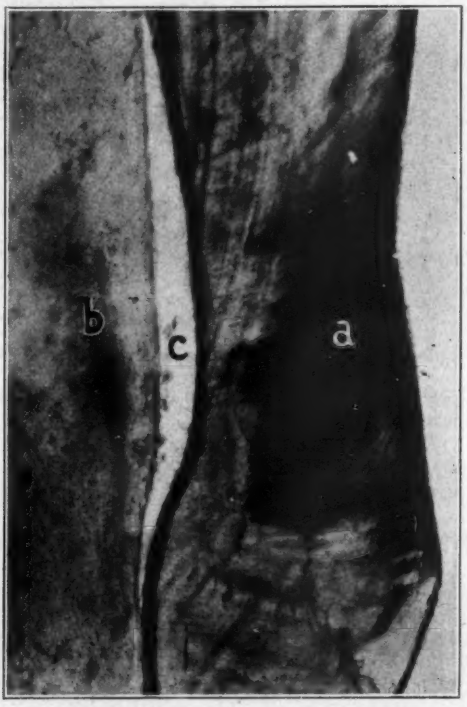

Fig. 12.—Ground section through tooth with intact discolored enamel surface. The discoloration (a) reaches not quite to the dentino-enamel junction at the level of this section. Considerable shrinkage of the dentin is shown at b, with only a trace of yellow discoloration. The space produced by shrinkage (c) is empty. The bordering lines of enamel and dentin are smooth without defect.

Figure 10 is the ground section through a tooth with an intact enamel surface. The frontal caries (a) has reached the dentin. The lines of Retzius in the carious area are accentuated, apparently by increased invasion into the

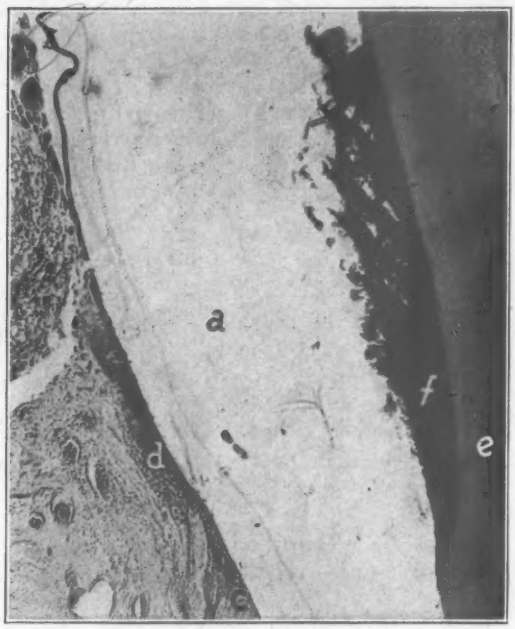

Fig. 13.—Decalcified section. Enamel dissolved by the acid used in the histologic technic (a). The cuticle of the enamel (b) is united with the gingiva (c) at the base of the crevice. Deposition of necrotic material at the surface of the enamel is indicated at d. In a depression caused by shrinkage of the dentin (e) is coagulated lymph (f).

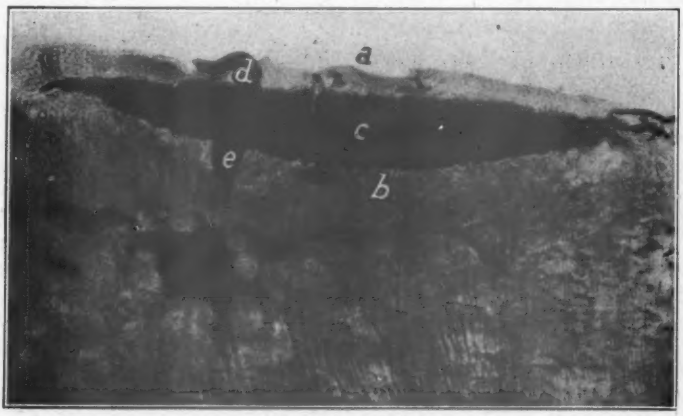

Fig. 14.—Decalcified section; enamel (a) dissolved; depression in dentin (b) caused by shrinkage; coagulated dental lymph (c) filling in depression. One extension of this necrotic material (d) goes into the enamel; another (e) penetrates the dentin.

less calcified strips. There is a trace of shrinkage in the dentin. The black spot (b) is yellow, the white homogeneous area (c) is partly decalcified (transparent dentin of first degree). The dark strand leading to the pulp (d) is the hypercalcified opaque tract. Such a condition may show up in a very good x-ray picture.

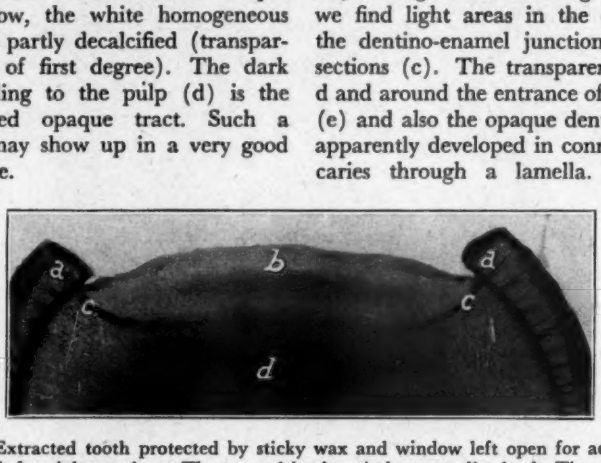

Fig. 15.—Extracted tooth protected by sticky wax and window left open for action of lactic acid (pH 4.6) for eighteen days. The enamel in the window was dissolved. The dentin shrunk away from the protected enamel margins (a). The superficial part of the shrunken dentin (b) shows homogeneity. Opaque strips (c) separate it from the normal dentin (e).

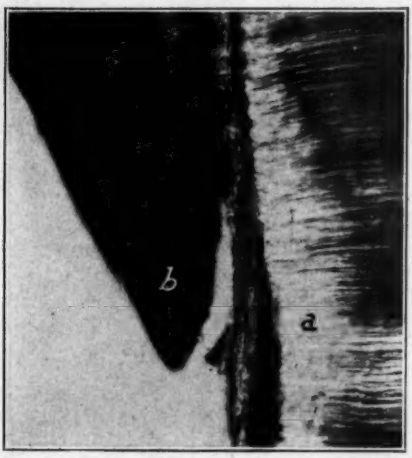

Fig. 16.—Longitudinal ground section through extracted tooth with chalky spot at cemento-enamel junction. The dentin (a) shrunk away from the enamel margin (b). Only traces of yellow pigmentation are to be found in the homogenized dentin. The chalky enamel margin is cloudy.

Figure 11 is the ground section of a tooth with a brownish depressed area at the enamel surface (a). The frontal caries (b) has already reached the dentin, causing a small shrinkage (d). Often, we find light areas in the dentin near the dentino-enamel junction in ground sections (c). The transparent dentin at d and around the entrance of the lamella (e) and also the opaque dentin (f) have apparently developed in connection with caries through a lamella. The light dentin around the shrinkage area is decalcified. The light dentin around the lamella is probably hypercalcified. A definite statement could only be made on the basis of grenz-ray examination.

Figure 12 shows the ground section through a brownish spot in a contact plane with an intact surface near a carious cavity. The dark area in the enamel (a) is pigmented brownish and does not reach the dentino-enamel junction in this plane. Here, a great amount of acid has apparently been produced, the dentin shrinking considerably (b) as a result. A space produced between dentin and enamel (c) appears empty. The bordering lines of enamel and dentin are smooth, not showing any defect. There are only traces of yellow pigmentation in the shrunken dentin. The incremental lines in the carious enamel are accentuated as in Figure 10.

Figure 13 is a decalcified section. The enamel (a) has been dissolved. The dentin (e) shows a depression caused by shrinkage from acid action. In the depression is a dark mass of coagulated dental

lymph (f). Small extensions of it penetrate the dentin at its apical end. The cuticle (b) indicates the outer borderline of the enamel with necrotic material at its surface (d). The base of the crevice is apparently at c.

Figure 14 shows similar conditions. The dissolved enamel (a) is separated from the dentin (b) by shrinkage of the dentin. In that area, there is an amorphous material (coagulated lymph) with extensions into the enamel (d) and the dentin (e). The carious process appears to spread from this focus in both directions.

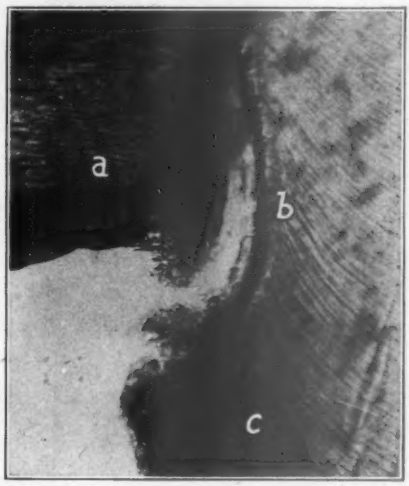

Fig. 17.—Ground section. The blunted enamel margin (a) is separated from the shrunken dentin (b). C, yellow pigmentation in apical portion.

Figure 15 shows an extracted tooth which was covered with sticky wax and a window left open to acid action. Lactic acid (pH 4.6) was allowed to act for eighteen days. The exposed enamel was dissolved. The dentin was decalcified and developed a transparent appearance (b). It shrunk and left the enamel borders (a) unsupported. At the borderline between decalcified and normal dentin (d), opaque lines (c) developed, apparently by addition of calcium salts from the declacified parts.

Figure 16 shows a longitudinal ground section through an extracted tooth with chalky enamel at the cemento-enamel junction. The enamel appears opaque. No loss of enamel substance in grinding indicates the strong condition. The enamel edge (b) has lost its dentinal support by shrinkage of the dentin (a). The shrunken dentin looks more homogeneous (transparent) than the more opaque neighboring dentin. The cleft between enamel and dentin seems to continue as a slit. Yellow pigmentation was to be found in traces in the dentin. Pigmentation of the enamel could not be definitely determined.

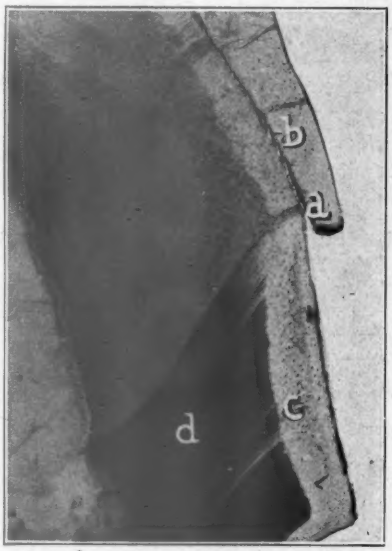

Fig. 18.—Window at cemento-enamel junction left open to acid action (pH 4.6) for twenty-six days. On the thirteenth day, tricalcium phosphate and an extracted tooth were added. The enamel margin was blunted (a). The adjoining enamel (b) was separated from dentin by a slit. Shrunken decalcified dentin (c) was homogenized. There is an opaque tract (d) going to the pulp canal.

Figure 17 shows a more advanced

condition. The enamel edge is missing. The blunted enamal edge (a) is separated from the dentin (b) by shrinkage of the dentin. One can find only traces

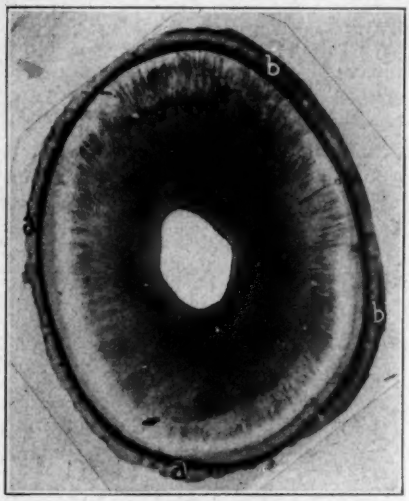

Fig. 19.—Horizontal ground section near the cemento-enamel junction through mottled tooth from fluorine-containing area. A belt of transparent enamel (a) runs along the surface. The part which shows some opaqueness (b) has an intact surface. In the other areas, the enamel was brittle.

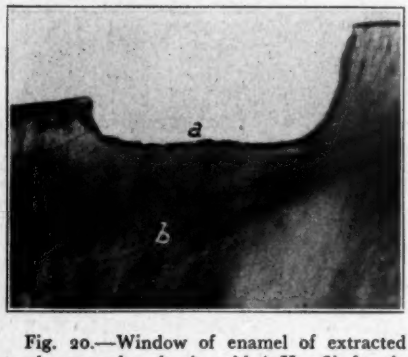

Fig. 20.—Window of enamel of extracted tooth exposed to lactic acid (pH 4.6) for six days. A portion of enamel (a) was clearly cut out. b, intact enamel.

of yellow pigmentation (b). Heavy pigmentation is present (c). The necrotic deposit at the surface of c indicates that the enamel edge was missing before extraction of the tooth. One small spot in the pigmented dentin has lost its histologic structure and is necrotic.

Figure 18 shows the result of acid action at the cemento-enamel junction

Fig. 21.—Enamel of extracted tooth exposed to action of lactic acid (pH 4.6) for three and one-half months. Plenty of tricalcium phosphate was added to the acid. The intact enamel surface at a was cloudy. b, normal enamel.

on an extracted tooth. The whole tooth was covered with sticky wax and a round window was left open at one place on the cemento-enamel junction. The tooth was put into a bottle with 7 cc. of lactic acid (pH 4.6) for twenty-three days. Midway of the experiment, plenty of tricalcium phosphate was added. A longitudinal ground section was made through the window. The enamel edge is missing. It was apparently dissolved

in the acid. The blunt end of the enamel (a) shows a considerable separation from the dentin. The more coronal parts of the enamel (b) are separated from the dentin by a slit (Fig. 16) caused by shrinkage of the dentin, which becomes evident on examining its contour. The peripheral part of the shrunken dentin (c) is transparent and apparently decalcified. In the deeper layer of the dentin, an opaque area (d) is to be seen which extends to the pulp cavity. That opaqueness could be washed out and was apparently caused by entrance of tricalcium phosphate powder into the tubuli. (Compare opaque tract, Fig. 10.)

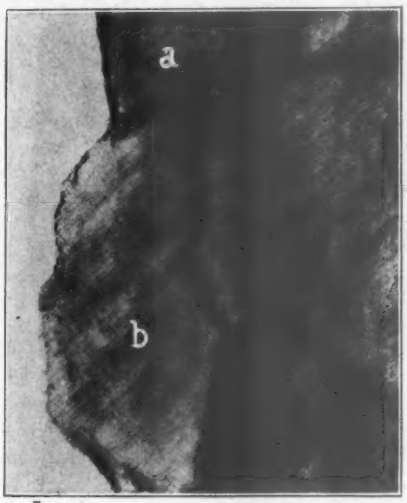

Fig. 22.—Tooth with brownish spot subjected to 0.1 per cent lactic acid for one month, with acid changed every day. a, transverse striation. The carious area (b) was more resistant to acid action.

Figure 19 is a cross ground section of a mottled tooth from a fluorine area with 4 parts per million fluorine in the water. The young patient was born and reared in this area. The section runs near the cemento-enamel junction where the enamel is thin. A belt of transparent enamel travels the whole circumference. At the left side (a), it is quite transparent. On the right side (b), the enamel is more opaque, especially in the upper portions. On the left side, the surface is irregularly broken. On the right side, between the two (b), it is intact.

Figure 20 shows the result of lactic acid action (pH 4.6) on a limited exposed area of enamel for six days. A part of the enamel (a) has been smoothly cut out while the remaining enamel (b) is intact.

The tooth in Figure 21 was exposed to lactic acid of the same concentration. Sufficient tricalcium phosphate was added to the acid and the mixture was renewed every four days. The experiment continued for three and one half months. The result is cloudy enamel (a) at the surface and intact enamel (b) in the deeper parts, with no dissolution of enamel substance.

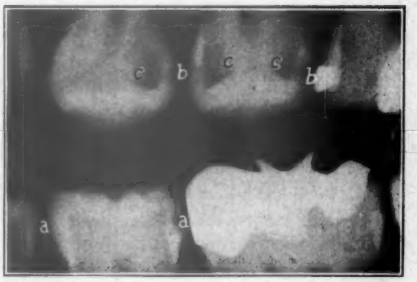

Fig. 23.—Bitewing film. On the distal side of the lower premolars (a) a defect in the enamel is evident. On the distal side of the upper first premolar and both sides of the second premolar (b), there are shadows in the enamel. There is an extensive shadow in the dentin (c) at the dentino-enamel junction.

Figure 22 shows the ground section of a tooth which was decalcified in 0.1 lactic acid for one month. The acid was renewed every day. The tooth surface became chalky in every case of decalcification in the test tube. The superficial layer of the enamel was dissolved. At a, we see transverse striation, the result of acid action. There was a brownish

area of frontal caries on the tooth. It resisted acid action (b) more than the neighboring enamel; which indicates that carious enamel is more resistant to acid action.

Figure 23 is the enlargement of a bitewing film. The upper first premolar shows a light cone-shaped area in the enamel on the distal side (b). The enamel surface appears to be intact. The cone is produced by a partial decalcification. About half of the enamel appears to be intact in its dentinal part. In the dentin, a shadow extends over the greatest distal part including a part of the occlusal plane (c). The same condition exists on the mesial surface of the second premolar. On the distal aspect of this premolar, the shadow in the enamel is greater, but the surface of the enamel seems to be intact too. A strip of roentgenologically intact enamel still separates the shadow in the enamel from that in the dentin. On the distal surfaces of the lower premolars (a), there are roentgenologic defects in the enamel which are darker than those in the upper premolars. The dentin in both lower teeth is intact. The mesial surface of the lower second premolar shows how intact enamel and dentin look and may serve for comparison.

(To be continued)

USE OF SILICOPHOSPHATE CEMENTS FOR ANTERIOR RESTORATIONS

JESSE GOODMAN, D.D.S., New York, N. Y.

THE silicate cements, also referred to as synthetic porcelains, were originally introduced to the dental profession as substitutes for metallic fillings in the anterior teeth. Some dentists even used them in posterior restorations. At first, the profession readily accepted this material because of its esthetic appeal, but, with clinical experience, its actual practical value was questioned. Failures, from pulpal death, solubility, shrinkage and color instability, were not infrequently encountered.

Articles in dental literature pertaining to the clinical disadvantages of silicates led to extensive research and experimentation to improve their composition and physical properties. The Research Commission of the American Dental Association set up exacting specifications to which all recognized silicates had to conform.

Granted that the silicates are greatly improved in their present form, articles still appear concerning their disadvantages. The directions for mixing supplied with the various brands insist on adherence to a rigid technic, but observance of these instructions does not obviate clinical difficulties. In all probability, the fault is not in the technic, but rather in the chemical composition and physical properties of this filling medium. The chemical composition is no secret, although the exact nature of the setting reaction is not known.

The setting of zinc phosphate cements results in the formation of crystalline compounds. It is assumed that the setting of silicates is different, since it is more of a colloidal nature, similar to that of a silica gel. Microscopic examination reveals particles of the cement powder embedded in a silicic acid type of gel matrix. Careful investigators have advanced the opinion that the gel binder

or matrix material is responsible for most of the inferior qualities of silicate cements.

Some years after the appearance of the first silicate cements for anterior teeth, new cements of a modified silicate type were introduced for posterior teeth. The powders consisted of combinations of silicate and zinc phosphate; silicate and zinc oxide together, or in combination with magnesium oxide. These combinations are often referred to as silicophosphates or silicate-zinc phosphate combination cements. Their purpose was to act as a substitute, to a certain extent, for gold and amalgam in posterior occlusal restorations and also to discourage the use of anterior silicate cements in posterior teeth. It is reasonable to assume that their physical properties must be superior to those of the silicates for anterior teeth; otherwise, they would not have been recommended for occlusal and compound fillings.

This assumption is justified by a statement in a circular of the National Bureau of Standards entitled "Physical Properties of Dental Materials" (C443), which reads as follows: "Where a cement is desired which has some characteristics of both silicate and zinc phosphate types, this kind of mixture appears to have in general superior physical properties."

Unfortunately, little independent research has been done on the silicophosphate cements. Furthermore, the published results do not give the true physical properties of this material from a clinical point of view. One of the published tests was performed at the National Bureau of Standards, according to the American Dental Association Specification No. 9 for Dental Silicate Cement, notwithstanding that silicophosphates are different in many respects from true silicates. The tests were made with samples of silicophosphate cements mixed to the same standard consistency as the silicate cements.

The method used to obtain this standard consistency was as follows: By repeated tests, trial amounts of the cement powder were added to 0.4 cc. of cement liquid. Then 0.5 cc. of the mixed, but unset cement was placed on a flat glass plate. Two minutes after the mix was started, another flat glass plate, with a weight resting on it, was carefully placed on the soft cement. The combined weight of the top plate and the weight, which was 2,500 gm. (5.51 pounds), caused the mixed cement to slump into a disk-shaped mass. Ten minutes after the starting of the mix, the glass plates were separated and the diameter of the cement disk was determined. Trials were continued until the diameter of the disk was 25 ± 1 mm. Once this was arrived at, the procedure was repeated three times, using 0.4 cc. of the liquid in each trial. The average weight of the powder used in three such tests was accepted as the amount of powder necessary to produce a mix of standard consistency.

Whether this consistency can be taken as one displaying the optimum physical properties of the cement is answered in the Bureau of Standards Circular C443:

> Let it be emphasized that this may not be the optimum consistency which will produce the best physical properties. . . . Objections have been raised to the testing of all cements at a standard consistency. The makers of these cements want their products tested at the consistency they recommend, which is invariably heavier than the standard.

The Bureau responded to this complaint by offering five reasons for their selection of a standard consistency for testing purposes; one of them being that "if the consistency requirement in the specification was changed so that a heavier mix was required, the resulting properties of all the cements would be improved, and this would necessitate a general upward revision of the specification requirements." It added that this,

however, would not make the specification more restrictive than it now is."

The choice of one standard consistency for all the silicate and silicophosphate cements may be convenient for laboratory testing methods but, for practical purposes in the mouth, the thickest permissible mixture of the particular cement should be utilized.

From a clinical point of view, the importance of powder to liquid ratio must be taken into consideration. It is admitted in the Bureau of Standard's circular C433 that the addition of more powder than is required for the standard testing consistency (1.4 gm. of powder to 0.5 ml. of liquid) considerably improves the physical properties of the mix. The addition of only 0.2 gm. of powder (one-seventh of the quantity used) will make a marked difference. This small increase in powder to liquid ratio will impart greater strength and decrease the solubility and shrinkage of the set mix. Furthermore, the thicker mix will contain relatively more powder and less free binder or gel matrix. In consideration of the fact that the binder or gel matrix is assumed to be responsible for many of the disadvantages of the silicates, it is obvious that the less free gel present, the stronger the cement.

There is a limit to the amount of powder that may be successfully added to the liquid. If more powder is incorporated in the mix than can be chemically attacked by the liquid, the presence of the uncombined powder particles will considerably weaken it. It is therefore inadvisable to mix the ordinary anterior silicates to the same thick putty-like consistency as recommended for the posterior silicophosphates. Likewise, the silicophosphates must not be mixed to the consistency recommended for silicates; otherwise, their true physical properties will be lacking. A proper mixture of silicophosphate presents a much drier surface with a less liquid consistency than that of a silicate, and is therefore less toxic. This fact is corroborated by a statement in the Bureau of Standard's Circular C433 concerning the acidity of silicate cements:

> This means that when the plastic cement is first introduced into the cavity, its acidity is relatively high, and it is probably at this time that the greatest injury to the pulp is liable to occur. This is especially true if the dentine has been severely dried and so has rapidly absorbed some of the liquid portion of the very acid unset cement. The need of varnishing the cavity floor and walls to prevent or retard the penetration of such acid substances toward the pulp, is very evident.

In addition to the decrease in acidity resulting from the drier surface of the silicophosphate mix, the liquid component is less acid than that of a silicate. By actual test with nitrazine paper (Squibb), the silicophosphate liquid was definitely less acid than the liquid furnished with a popular brand of silicate cement.

I have successfully used one brand of silicophosphate ever since its introduction to the dental profession in America. This material was first used primarily in posterior teeth when non-metallic fillings were indicated because of the probability of a galvanic effect, and also to replace metallic fillings in teeth with hypersensitive pulps which responded to thermal shock even with an intervening cement lining. Although a protective cavity varnish was first placed over the cavity surface, it was soon evident that its use was unnecessary. When it was found that this material was bland and non-irritating if mixed to the proper consistency, it was used as a filling medium in deep-seated cavities in deciduous teeth, with gratifying results.

It seemed plausible that if this material proved so satisfactory in posterior teeth, it might be valuable for anterior restorations.

It will be admitted that anterior silicophosphate fillings do not appear so translucent as the anterior silicates when first placed. Also, owing to the increased

density of the silicophosphates, the shade appears lighter. However, it is an undeniable fact that the anterior silicates have a tendency to discolor in the mouth. Sometimes, they turn a dingy brown in a relatively short time after insertion. Such is not the case with silicophosphates. A careful observation for almost twelve years proved this to be so. The original shade of the silicophosphate selected when the fillings are inserted remains constant without any noticeable change.

Controls were made with silicate anterior fillings and silicophosphate anterior fillings in the same mouth. After a relatively short time, the silicates were quite discolored and required replacement. The silicophosphates retained their original color and were of more pleasing appearance than the silicates.

Further control observations were made to determine whether the silicophosphates were more durable and less irritating than silicates. In mouths where daily care was apparent, the longest time that silicates endured was five years. There were many instances wherein replacements were required in less than two years. The silicophosphates, on the other hand, remained serviceable for from eight to ten years.

It is very likely that some members of the profession may not be entirely in accord with the claim made regarding the durability of the silicates. This, however, is the consensus of opinion of many careful practitioners and numerous research investigators. It is further evidenced in a report, published by G. C. Paffenbarger in 1940 and based on the findings of a group of practicing dentists under the direction of the American Dental Association Research Fellowship at the National Bureau of Standards. In this report, it is claimed that the average life expectancy of a silicate cement filling is 4½ years.

It was also noticed that a zinc oxide and eugenol cement lining or a protective cavity varnish was required in most deep involvements and also in cavities with sensitive dentin, if anterior silicates were used. This was not necessary with the silicophosphates.

Here again, doubt may arise regarding the toxic properties of silicates. It is true that the profession is still unsettled on this question. A fair appraisal of the diversity of opinion on this point may be based on the following statement appearing in the Bureau of Standard Circular C433:

> To illustrate how far apart professional opinions are regarding the toxic effects of silicate cement on the pulp, one has only to review the replies to the following question submitted to a group of 106 dentists who were cooperating with the American Dental Association Research Commission in the study of silicate cements. Question: "Do you believe that the silicate cements are more irritant to the pulp than are other restorative materials?" Approximately 45 per cent of the cooperators replied, "No," 45 per cent replied, "Yes," and the remaining 10 per cent were "undecided."

It is my contention, based on clinical and experimental experience with silicophosphates, that this filling material is ideal for anterior restorations. It is true that it is inferior to the silicates esthetically, but how many of us have seen anterior amalgam and plugged gold restorations remain serviceable for more than fifteen or twenty years? These materials are definitely more objectionable, in appearance, than silicophosphates.

It remains for us to determine whether appearance shall be given preference over health. Our patients must likewise be enlightened on this most important decision.

The possessor of a false limb or a glass eye is fully aware of the degree of artificiality present, but is, nevertheless, agreeable to its use. Why cannot we convince our patients of the value of sacrificing a certain degree of beauty for the good of the tooth?

222 West Eighty-Third Street.

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NOVEMBER 1 *Editorial Department* 1944

DENTISTRY IN THE ARMY

SOME months ago, the War Department announced discontinuance of the Specialized Training Program so far as dentistry was concerned, presumably because there has been a material reduction in the expected dental requirements of the Armed Forces, particularly the Army.

When this action was announced, steps were immediately taken by the American Dental Association to induce a reconsideration of the action or, rather, to defer its application, and protests were entered from the President of the American Dental Association, the War Service Committee and a special committee of the American Association of Dental Schools.

The plea for continuation of the program was based on the dental health needs of the Armed Forces, the disruption of the general educational program for dentistry and upon what appears to be an injustice to the young men who were drafted into the army service by way of the dental health service. These protests have proved of no avail, but have called from the War Department a reaffirmation of its intention to pursue its course of abandonment of the student program and to reaffirm its intention to materially reduce the dental health service of the Armed Forces as represented by the Dental Corps.

The recourse offered to the students involved is discharge from the

Army or assignment to the Medical Department as enlisted men; or they may be discharged from the Army and deferred from military induction in order to continue their professional course, provided they have letters of acceptance and will enter accredited dental schools not later than December 31, 1944.

When the War Department adopted this new policy, we were of the opinion, or rather hope, that it would be only temporary, that the need for dental service would demand a return to the original program, but it is evident now that the War Department is determined to abandon the Dental Student Training Program and that the educational system of dentistry is left to its own devices to unscramble the confusion caused by experimenting with this Army program.

The War Department states specifically that there is no need for more dentists in the Army and that they do not want any more.

In keeping with the constancy of our patriotism, it appears that the dental profession will be compelled to accept this dictum with as much grace as possible, and as another exemplification of the trueness of the famous characterization of war by General Sherman.

The one bright hope of some good from this ill wind is that it may eventuate in increasing the number of dentists in civilian practice. In fact, the government has intimated its hope that many of these students will continue their dental course and thus augment the output of civilian graduates into dentistry.

Additional evidence of the oversupply of dentists in the Army is afforded by the expressed intention of the government to begin soon to discharge dentists from the service.

It would seem that the War Service Committee must have sensed some such turn in the course of events when the recent questionnaire was sent out soliciting an expression of the wishes and desires of the men now in the service, when they are discharged.

Incidentally, a casual preliminary survey of the replies to the questionnaire made by the chairman of the War Service Committee, C. Willard Camalier, furnishes some interesting light on the attitude of the men in the service toward their profession. Many of them evince a definite desire to pursue postgraduate and refresher courses when discharged; many of them hope for and expect financial and professional aid in reëstablishing practices; many of them anticipate the availability of surplus dental equipment and supplies to the returning officers, and many expect more favorable provision for reciprocity in dental licensure. Practically none of them expresses the desire to abandon dental practice—and this we take to be an encouraging step toward meeting the demands that will be

placed upon dentistry as a health service to the public.

We have had many anxious inquiries from men in the service and out as to the ultimate fate of those students in the midst of their dental education, yet scheduled to be discharged from Army service before the end of the year. We have answered this question in the MID-MONTHLY, but, in the desire to leave no doubt as to the possible fate of these men, we repeat again our interpretation of the provisions of the Readjustment Act of 1944, pertaining to this question.

Any student now in the Army Specialized Training Program has two elections:

He can elect to apply for a discharge from the service and continue his studies on a civilian basis without any aid from the Government. We are informed that in such cases, under recent rulings, the student will be deferred from military service for the duration.

He can elect to give up his dental training for the time being and be transferred to the Medical Corps and continue in military service as a Medical Corpsman on a non-commissioned basis. In the latter case, if he serves ninety days or more prior to the termination of the present war or if he shall have been discharged because of a service-incurred disability, he shall be eligible to the educational benefits provided in the Servicemen's Readjustment Act of 1944, more commonly known as the G. I. Bill. (JOURNAL, October 1, 1944, page 1389.)

We have also had many inquiries from men in the Dental Corps as to the possibility of discharge of men now in the service, and, in answer to that inquiry, we understand that beginning in December, those of 38 years and over will be discharged at the rate of approximately 100 per month. The Servicemen's Readjustment Act provides for the educational rehabilitation of those men who wish to avail themselves of the benefits offered by the bill—refresher courses and extension of their educational study at the expense of the government.

When the War Department first announced discontinuance of the Specialized Training Program, we were apprehensive lest too many men should abandon the intention of completing their dental education and that the inflow of new graduates into the profession would be materially inhibited.

Our latest information on the subject, however, indicates that the large majority of those now in course will continue their studies to completion and enter the ranks of dentistry. If such continues to be the case, the only regret that we can have as to this sally of the Government into our educational field concerns the disturbance of a dental educational system that has been tried and found efficient through the past decade.

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BUREAU OF PUBLIC RELATIONS

EMERGENCY MATERNAL AND INFANT CARE DENTAL TEST PROGRAM

LEON R. KRAMER,* D.D.S., M.S.P.H., Topeka, Kan.

REQUESTS for emergency dental care are being received by state Emergency Maternal and Infant Care Program directors.

Many pregnant wives of service men in the lower four pay grades, and who are stranded near camps, have not received their checks from the government, or, for some other reason, are unable to provide emergency dental care for themselves. The only means open to them is an appeal to the charity of local dentists or application for service in welfare clinics. In consideration of service men's pay in the lower four pay grades, they and their dependents all represent a low income group.

To meet this situation, "the dental profession," in the words of C. Raymond Wells, President of the American Dental Association, "should make some provision whereby the activities of our profession can be correlated with the Emergency Maternal and Infancy Care Program."

This was the background supplied to me as director of the Division of Dental Hygiene, Kansas State Board of Health, by Captain Wells and others, before I attended the Advisory Committee Meeting on Maternal and Child-Health Services, Children's Bureau, U. S. Department of Labor, of which I am a member, representing, with Lon W. Morrey, of Chicago, the American Dental Association in maternal and child health activities.

* Director, Division of Dental Hygiene, Kansas State Board of Health.

APPROVAL OF PRINCIPLES OF PLAN FOR PROVIDING EMERGENCY DENTAL CARE FOR APPROVED EMIC CASES

At the meeting of the Advisory Committee on Maternal and Child Health Services, Children's Bureau, U. S. Department of Labor, held in Washington, October 21, 1943, Martha M. Eliot, associate chief, Children's Bureau, presented the following problem to me as a director of a state dental health program and as a representative of the American Dental Association on this committee. "Assume, where the patient is in need of emergency dental services, such care might be given by dentists in a consultant capacity."

Edwin F. Daily, director, Division of Health Services, Children's Bureau, also requested me to attempt to work out a plan along the lines suggested above.

Since that time, the reactions of dental officials on national and state levels have been surveyed. These officials include the President of the American Dental Association, the Council on Dental Health, officers of the American Association of Public Health Dentists and officials of the Kansas State Dental Association and the Kansas Council on Dental Health. All were in accord on the following points:

1. The dental profession should correlate its activities with the Emer-

gency Maternal and Infancy Care Program.

2. Dental services should be limited to those designated as emergency services.
3. These services should be limited to cases approved by the Emergency Maternal and Infancy Care Program, at the present time.

Dental Association and May 4, 1944, the Kansas State Dental Association approved the principles of this program.

Plans, together with details, relative to procedure and implementation were presented April 10, 1944, to the Advisory Committee of the Kansas Emergency Maternal and Infancy Care Program, of which Drs. Lattimore, Belknap and Loveland are members. The principles of correlating the activities of the dental profession in the Kansas Emergency Maternal and Infancy Care Program in the capacity of consultants for providing emergency dental care for maternity cases and of surveying the possibilities of expanding the dental plan

KANSAS STATE BOARD OF HEALTH

FORM M-2-1 Dental
M-2-2
(check one) Division of Maternal and Child Health

AUTHORIZATION FOR EMERGENCY DENTAL CARE

Do not write in this space

Case No.....

Authorization No.....

Effective Date.....

Emergency Maternity and Infant Care Program for Dependents of Servicemen

To.....D.D.S. Address.....

Patient.....Address.....

This is to certify that the Kansas State Board of Health will assume responsibility for payment for the following services to be provided the above-named patient.

Emergency Dental Care.....

All care or services authorized will be paid for by the state board of health according to the rates stated in the U. S. Veterans Bureau Fee Schedule.

The patient and/or dentist indicated on application will be notified that this authorization has been made effective as of.....194.....

Signature.....Director
Division of Maternal and Child Health, Kansas State Board of Health

Note: Invoices for services, when submitted to the director, Division of Maternal and Child Health, ogether with the final reports (M-3 Dental) constitute the basis for payment of all services provided after date of application. It is not necessary to return this authorization when submitting the final report.

Amount authorized: M-2-1 variable amounts up to $5.
(check one) M-2-2 variable amounts up to $5. Amount paid.....Date.....

Note: M-2-1 is the original authorization for amounts up to $5. If $5 does not cover the cost of care or if subsequent care is needed, dentist will explain case and request Form M-2-2, which authorizes additional amounts up to $5.

STATE OF KANSAS

The reaction to a questionnaire relative to the initiation of this program in Kansas, sent to officers of the Kansas State Dental Association and to the Kansas Council on Dental Health, was 100 per cent favorable. May 3, 1944, the Executive Council of the Kansas State

KANSAS STATE BOARD OF HEALTH

FORM M-3 Dental

Division of Maternal and Child Health

DENTIST'S REPORT OF DENTAL CARE

Do not write in this space

Case No.....

Authorization No.....

Effective Date.....

Emergency Maternity and Infant Care Program for Dependents of Servicemen

Patient's name..... Address.....

Dental services: These services shall consist of emergency dental care for the purpose of controlling or reducing pain (such as toothache) and/or infection (such as acute Vincent's infection, or "trench mouth") in the hard and/or soft tissues of the mouth.

The authorization Form M-2-1 Dental authorizes the payment for emergency dental services by the Kansas State Board of Health up to and including $5 to licensed dentist practicing in Kansas. Should additional services be needed, the dentist shall give an explanation of the case and shall send a request for a supplementary authorization, Form M-2-2 Dental, for additional services up to and including $5 to the director, Division of Maternal and Child Health, Kansas State Board of Health. Ten dollars ($10) is the limit that can be paid for emergency dental care for one case.

The U. S. Veterans Bureau Dental Fee Schedule, approved for contract service by the American Dental Association and the Kansas State Dental Association, for Emergency Dental Services, follows: Please indicate number and type of service rendered this case, for which payment is authorized by the Kansas State Board of Health.

Number of Services	Fee Schedule		Amount
	Emergency treatment—palliative	$1	
	Simple extraction with local anesthesia	$2	
	Postoperative treatment	$1	
	Roentgenogram—single	$1	
Minutes	Prophylaxis (in conjunction with treatment such as for acute Vincent's infection) at rate of	$4 per hour	
	Other		
		Total	

Authorization for emergency dental care will be effective from the date stated on authorization Form M-2-1 Dental, to the end of the sixth week after the date of delivery. Dentists should make a careful check on effective dates.

Mail to:—Director, Division of Maternal and Child Health, Kansas State Board of Health, Topeka, Kan., within thirty days after the service has been rendered.

I hereby certify that I have not received payment for any authorized service indicated above from the patient or other sources.

Record completed (date)..... 194.....

Signed..... D.D.S.

Address.....

to include dental care for dependent children of service men in the lower four pay grades, met with their approval. They recommended that these principles be submitted to the Kansas State Board of Health for approval, an action which would give clearance to the submission of the plan or plans devised through the coordinated efforts of dental officials and the Kansas Emergency Maternal and Infancy Care Advisory Committee, in their final form, to the Director, Division of Health Services, Children's Bureau, U. S. Department of Labor.

Under these conditions, the following statement was submitted for consideration by the Kansas State Board of Health:

That the Kansas State Board of Health accepts doctors of dental surgery or doctors of dental medicine licensed and practicing dentistry in Kansas, in the capacity of consultants in the Kansas EMIC Program and approves the principles of supplying emergency dental care for approved EMIC cases and of giving future consideration to the provision of dental care to dependent children of service men in the four lower pay grades.

This statement was presented in the form of a resolution and was passed April 13, 1944.

The Kansas plan for EMIC, page 6, paragraph 7, indicates that thought has been given relative to initiating a dental care program after July 1, 1944. The paragraph that records the favorable consideration given to this item by the Kansas Program Committee follows:

ADMINISTRATION POLICIES, EMERGENCY MATERNITY AND INFANT CARE PROGRAM
(KANSAS)

Section III.—Application and Authorization for Care, Item B—Authorization for Care, Page 6—Paragraph 7:

It is recognized that dental care will be needed by many patients requesting services under these plans, but, in view of the limited funds available at present, no provision will be made for the payment for dental services from Fund E during the remainder of the current fiscal year. (Ending July 1, 1944)

Therefore, the ground work has been prepared whereon a state program for the purpose of supplying dental care for Emergency Maternal and Infancy Care cases can be tested. The program will be put into operation October 1, 1944.

KANSAS EMIC DENTAL PROGRAM

I. Purpose:

The purpose of this program is to supply emergency dental care to pregnant wives of service men in the lower four pay grades, whose cases have been approved by the Kansas Emergency Maternity and Infant Care Program.

II. Type of Service:

These services shall consist of emergency dental care for the purpose of controlling or reducing pain, such as toothache, and/or infection, such as acute Vincent's infection (trench mouth), in the hard and/or soft tissues of the mouth.

III. Period of Coverage:

These services shall be available to approved cases from the date of authorization for dental care to the end of the sixth week after the date of confinement.

IV. Choice of Dentist:

The patient may exercise her choice of dentist, located in Kansas, to render dental service.

V. Services Rendered:

Only dentists, licensed and practicing in the state of Kansas and approved by the Kansas State Board of Health, will be authorized to perform dental services, for which payment will be made from Kansas EMIC funds, through the Kansas State Board of Health.

VI. Fee Schedule:

Payment for dental services rendered will be in accordance with the U. S. Veterans Bureau Fee Schedule.

VII. Administration:

Whereas the principles, policies and procedures will have been checked and approved by official health organizations involved in the program, the actual mechanics of execution shall be carried out as an added

KANSAS STATE BOARD OF HEALTH

FORM M-1 Dental

Division of Maternal and Child Health

APPLICATION FOR EMERGENCY DENTAL CARE

Do not write in this space

Case No.....

Authorization No.....

Effective Date.....

Emergency Maternity and Infant Care Program for Dependents of Servicemen
(Void 6 weeks after date of delivery)

Part One — Patient's Application

Patient's name.....Date of birth.....
(Last) (First) (Middle)

Present address.....
(Street) (City)

.....Telephone No.....
(County) (State)

Name of husband.....Serial No.....
(Last) (First) (Middle)

Husband's mailing address.....

Branch of service.....Rank or rating.....
(Army, Navy, Marine Corps, Coast Guard) (If sergeant, specify type)

I herewith request emergency dental services, when and if needed under the Kansas EMIC program from the date of signature until six weeks after date of delivery.

I understand that the Kansas State Board of Health will make payments for all authorized services provided, and that no additional payment may be made by me or by other persons in my behalf for such services.

Date..... Applicant's signature.....

If patient desires to apply for emergency dental care, she shall present this application to the dentist of her choice for his signature and mail this application at once to:

Division of Maternal and Child Health, State Board of Health, Topeka, Kan.

Part Two — Dentist's Statement

Dental Services: These services shall consist of emergency dental care for the purpose of controlling or reducing pain (such as toothache) and/or infection (such as acute Vincent's infection, or "trench mouth") in the hard and/or soft tissues of the mouth, when and if needed.

I request authorization to provide under the EMIC program the necessary dental emergency services for this patient during pregnancy and six weeks postpartum beginning at date of signature and agree that I will accept payment only from the Kansas State Board of Health for such services.

Date of signature.....

Dentist's signature.....Degree.....

Address.....
(If clinic dentist or official, give name of clinic or hospital)

PLEASE SEE THAT THIS APPLICATION IS MAILED AT ONCE

service in the EMIC program and handled in routine fashion.

VIII. EMIC Dental Committee:

Disposal of statements for dental services rendered by dentists and/or matters pertaining to special or questionable cases, will be referred to a committee composed of four dentists, appointed by the president of the Kansas State Dental Association, of which committee, the director of the Division of Dental Hygiene shall be a member. This committee will be designated the "EMIC Dental Program Advisory Committee." The work of this committee as related to the EMIC program will be with and through the director of the Division of Dental Hygiene, and this committee shall act in an advisory capacity to the director of the Dental Division in all matters pertaining to the launching and execution of the dental program. This committee shall, when needed, confer with the EMIC medical committee.

IX. Procedure:

A. All maternity cases authorized on and after October 1, 1944 by the EMIC director will be sent an application for emergency dental care. At the patient's option, she may fill out the application and return it to the Kansas State Board of Health. She will have properly filled out the application form and secured the signature of the dentist of her choice. (Form M-1 Dental.)

B. The State Board of Health will then send an authorization form for dental services to the patient and to the dentist of her choice. (M-2-1 Dental.)

b-1. The limit allowable for payment for dental services per case is $10. A case may need emergency care more than once during the prenatal period. Therefore, the $10 limit per case will be divided into two equal parts. The original authorization will authorize services up to $5. (M-2-1 Dental.) Since it is possible that $5 may not cover the cost of necessary care, a supplementary authorization (M-2-2 Dental) for an additional $5 may be granted, if needed. To receive the supplementary authorization, the dentist will be required to explain the case and make his request for the supplementary authorization. The supplemental authorization will be approved by the director of the Dental Division and/or the Dental EMIC Committee.

C. When and if dental services are needed the patient will go to the dentist designated on the application blank and procure the needed emergency service.

D. Upon completion of the service, the dentist will fill out the statement form (M-3 Dental) and mail it to the Kansas State Board of Health for payment, within thirty days after the service has been rendered.

E. Approval for Payment: The dentist's statements for ordinary services rendered will be approved according to the usual EMIC procedure. Questionable cases will be referred to and passed upon by the Dental EMIC Committee.

X. Memoranda on Expense of Program:

A. Data obtained from welfare programs in Kansas indicate that 15 to 20 per cent of indigent adults will apply for needed emergency dental service in one year.

B. Figures supplied by the Kansas EMIC director reveal that the average maternity case is covered for a period of about five or six months.

C. Limitation for fees per case will be based on that stated for minor and/or major surgery in the following items: EMIC Circular I, Chapter IV, Sec. C. Item 1, a-b-c, page 12.

c-1. The fee will be limited to $10 per case (certain exceptions possible). Anticipated actual cost per case $5 (based on 20 per cent of cases for a period of six months).

D. Commitment: The number of approved maternity cases in the Kansas EMIC program is approximately 800 per month, or 9,600 per year. Twenty per cent of 9,600 is 1,920 for a one year period or half this figure for a six-month period, which is 960. The commitment of funds needed, based on a $10 limit per case, would amount to $9,600 or $10,000 in round figures.

E. The expense of this program shall be paid from Fund E or Fund B.

XI. Implementation:

A. All practicing dentists in the state shall be notified by letter relative to the institution of this program. Included in this letter shall be a detailed explanation of all phases of the program and a copy of the U. S. Veterans Bureau Fee Schedule for dental services.

B. Forms for implementing program:

M-1 Dental: Patients application form.

M-2-1 Dental: Authorization for service up to $5.

M-2-2 Dental: Supplemental authorization for additional service up to $5.

M-3 Dental: Dentist's statement for services rendered to be sent to the Kansas State Board of Health for payment.

C. Exhibit of above forms are attached.

COMMENT

I. This program affords a test for determining the cost of supplying a minimal amount of emergency service. Later, it may be found advisable to expand the interpretation of emergency service to include other types of service.

II. Should dental services be rendered by employed dentists in clinics or hospitals, procedures shall conform to state practice acts. In no case, however, shall fees charged exceed those stated in the U. S. Veterans Bureau Fee Schedule. Public health clinics shall make no charge for these services.

COUNCIL ON DENTAL THERAPEUTICS

FEDERAL TRADE COMMISSION STIPULATION AND ORDER ON COLGATE DENTIFRICES

THE Federal Trade Commission has accepted from Colgate-Palmolive-Peet Co., Jersey City, N. J., a stipulation to cease and desist from disseminating radio, periodical and other advertisements which misrepresent the composition, properties and effectiveness of certain soaps, dentifrices and shaving creams that it manufactures and sells in interstate commerce.

February 3, 1944, the Commission issued a complaint[1] charging the respondent corporation with using unfair and deceptive acts and practices in commerce in connection with the sale of Palmolive Soap, Colgate Dental Cream, Colgate Tooth Powder, Palmolive Lather Cream, Palmolive Brushless Shave Cream and Concentrated Super Suds. After accepting the stipulation, the Commission closed the case growing out of the complaint without prejudice to its right to reopen the proceedings should the facts warrant.

With respect to its dentifrices, Colgate Dental Cream and Colgate Tooth Powder, the respondent corporation agrees to discontinue representing:

1. That the use of Colgate Dental Cream will impart to the teeth any radiance, brilliance, sparkle or like property beyond that inherent in the teeth of the individual user.

2. That the use of any of the respondent's dentifrices has any beneficial effect, except as a transitory mask, upon unpleasant breath odors other than those due to food particles or other matter in the mouth removable by the use of a toothbrush and the respondent's dentifrices.

3. That in all cases unpleasant breath odors due to decaying food particles in the mouth will be benefited by use of the respondent's dentifrices.

4. As "special" a foam or suds produced in the mouth of the user of any of the respondent's dentifrices due to the presence of soap; that is, a product of saponification of oils or fats.

5. That whether teeth are or are not properly brushed depends on whether

1. J.A.D.A., 31:555, April 1, 1944.

the respondent's dentifrices are or are not used.

6. By use of the word "ordinary" or other words of similar import, with reference to other dentifrices, that such other dentifrices are common or ordinary by comparison with the respondent's and are inferior thereto.

7. That tooth decay will be prevented by use of the respondent's dentifrice.

Stipulation and Order Closing Case 5126, September 5, 1944.

FEDERAL TRADE COMMISSION COMPLAINT ON ANACIN[1]

THE Anacin Co., Jersey City, N. J., and its advertising agency are charged in a complaint issued by the Federal Trade Commission with disseminating advertisements which misrepresent the therapeutic properties of Anacin Tablets and fail to reveal their potential danger to health when self-administered indiscriminately.

The advertising agency named in the complaint is the copartnership of Hill Blackett and Glen Sample, trading as Blackett-Sample-Hummert, Inc., 221 North LaSalle St., Chicago. It is charged that they have assisted in the preparation and dissemination of advertisements for Anacin Tablets.

Advertisements of Anacin Tablets inserted in newspapers and periodicals, pamphlets, circulars and other literature, the complaint alleges, have represented, expressly and by implication, that the preparation, because it contains several ingredients, is of a kind that a doctor would probably prescribe for persons suffering from various kinds of pains and is particularly valuable for that reason; that it can be depended on to relieve rheumatic pains, will prevent a cold if taken in time and shorten the duration of a cold, and reduce fever, and that there is no danger attendant upon its indiscriminate self-administration by the public generally.

The complaint charges these representations are false and misleading in the following respects:

It cannot be truthfully stated that Anacin Tablets is such a preparation as would probably be prescribed by physicians indiscriminately. Certain of its ingredients are more valuable in relieving definite types of pain than others. A doctor in prescribing an analgesic will determine the type of pain and prescribe the type of drug or drugs most effective for that particular type. A combination of several drugs, as in the respondents' preparation, does not necessarily make it more valuable or effective than a preparation containing only one. The product cannot be depended on to relieve the pains of rheumatism; it will not be effective in preventing or shortening the duration of a cold or in reducing fever, and there is potential danger to health in its indiscriminate self-administration by the public.

The complaint alleges that the respondents' advertisements are false for the further reason that they fail to reveal the harmful consequences that may result from the use of the preparation under the conditions prescribed in the advertisements and under such conditions as are usual or customary. The dosage recommended on the label of the container is: "One or two tablets with water. Repeat if necessary, one tablet in 3 hours. Do not take more than 5 tablets within 24 hours."

The respondents have represented that

1. J.A.D.A., 16:1121, June 1929.

the preparation will relieve the pains of rheumatism, headache, neuralgia and the menstrual period. In many cases, the complaint charges, these pains, as well as others which the layman cannot recognize as due to other conditions, will persist for an extended period and tend to recur after the palliative effect has worn off, which, in the case of the respondents' product, is not more than four hours for each prescribed dose. Because of these facts, the complaint continues, the customary condition in cases of persistent or recurrent pain is that there will exist a tendency for the sufferer to take more frequent and larger doses than prescribed, and the use of the preparation in a quantity exceeding the recommended dose or with greater frequency than recommended may be dangerous to health.

The respondents are granted twenty days to answer the complaint. Complaint 5213, September 9, 1944.

ACCEPTED DENTAL REMEDIES

The Council on Dental Therapeutics of the American Dental Association announces the inclusion of the following articles in the list of Accepted Dental Remedies.

DENTIFRICES*

Stratford Tooth Powder: Composition: See Sheffield Tooth Powder.

Manufactured by The Sheffield Company, New London, Conn.

Admission of a product to the list of Accepted Dental Remedies does not imply a recommendation. It means that the product and the methods by which it was marketed at the time of consideration were not found to be in violation of the Council's published rules. Accepted products are reconsidered periodically. The files of the Council contain information on many drugs and dental cosmetics. All information is available, upon request, and inquiries are welcomed. A postal card will bring a prompt reply.

TMC Tooth Powder: Composition: See Sheffield Tooth Powder.

Manufactured by The Sheffield Company, New London, Conn. Distributed by the May Department Stores Company, New York, N. Y.

Anesthetics—Local†

Procaine HCl 2%, Epinephrine 1:25,000. Each cubic centimeter is stated to contain procaine hydrochloride, 0.02 Gm.; epinephrine, 0.00004 Gm.; sodium chloride, 0.005 Gm.; sodium bisulfite, 0.0019 Gm., and distilled water. Marketed in cartridges, approximately 2.5 cc.

Procaine HCl 2%, Epinephrine 1:50,000: Each cubic centimeter is stated to contain procaine hydrochloride, 0.02 Gm.; epinephrine, 0.00002 Gm.; sodium chloride, 0.005 Gm.; sodium bisulfite, 0.0019 Gm., and distilled water. Marketed in cartridges, approximately 2.5 cc.

Manufactured by S. F. Durst & Co., Philadelphia, Pa. Distributed by Ethical Dental Products Company, New York, N. Y.

SULFONAMIDE COMPOUNDS‡

Sulfanilamide, U.S.P.—Sulfanilamidum (Sulfanilamid.) —Sulfanilamide, when dried at 100°C. for four hours, contains not less than 99 per cent of $C_6H_8N_2O_2S$.

*A.D.R. Ed. 10, p. 95.
†A.D.A. Ed. 10, p. 39.
‡A.D.R. Ed. 10, p. 192.

Sulfanilamide Sterile Powder, 1.0 Gm. ampuls and 5 Gm. vials.

Manufactured by E. R. Squibb & Sons, New York, N. Y.

Sulfathiazole, U.S.P.—**Sulfathiazolum (Sulfathiazol.)**—Sulfathiazole, when dried at 100°C. for four hours, contains not less than 99 per cent of $C_9H_9N_3O_2S_2$.

Sulfathiazole Sterile Powder, 5 Gm. Vials.

Manufactured by E. R. Squibb & Sons, New York, N. Y.

J. Bird Moyer Products

Acetone-Moyco: A brand of acetone, U.S.P.

Ammoniated Silver Nitrate-Moyco: A preparation containing 18% chemically combined silver nitrate.

Aromatic Solution of Phenol (Black's 1,2,3)-Moyco: A preparation containing oil of cassia 17%; phenol, 33%; methyl salicylate, 50%.

Chloroform-Moyco: A brand of chloroform, U.S.P.

Chromic Acid, 7% Moyco: A solution of chromium trioxide, U.S.P., 7% by weight.

Creosote-Moyco: A brand of creosote, N.F.

Dichloramine-T 5% Solution-Moyco: A 5% solution of dichloramine-T in Chlorcosane.

Glycerite of Phenol-Moyco: A brand of glycerite of phenol.

Iodine Disclosing Solution-Moyco: A preparation containing zinc oxide, 2.3%; potassium iodide, 2.3%; iodine, 7.7%; glycerin 49%, and distilled water.

Iodoform Powder-Moyco: A brand of iodoform powder, N.F.

Menthol-Moyco: A brand of menthol, U.S.P.

Monsel's Salt-Moyco: A brand of ferric subsulfate.

Oil of Wintergreen-Moyco: A brand of methyl salicylate, U.S.P.

Phenol-Menthol and Thymol-Moyco: A preparation containing phenol, 67.5%; thymol, 16.7%; menthol, 8.3%, and water, 7.5%.

Potassium Permanganate-Moyco: A brand of potassium permanganate, U.S.P.

Silver Nitrate 10% Solution-Moyco: A 10% solution of silver nitrate, U.S.P.

Tannic Acid, 25% Solution-Moyco: A solution of tannic acid, U.S.P. 25% by weight.

Thymol Iodide-Moyco: A brand of thymol iodide, U.S.P.

Trichloroacetic Acid 50% Solution-Moyco: A solution of trichloroacetic acid, U.S.P. 50% by weight.

Trichloroacetic Acid 25% Solution-Moyco: A solution of trichloroacetic acid, U.S.P. 25% by weight.

Tricresol and Formalin-Moyco: A preparation containing cresol, US.P., 42.8%; formaldehyde solution (40%), 44.6%, and ethyl alcohol, 12.6%.

Zinc Chloride, 50% Solution-Moyco: A solution of zinc chloride, U.S.P. 50% by weight.

Manufactured or distributed by the J. Bird Moyer Company, Philadelphia, Pa. No U. S. patents or trademarks.

Admission of a product to the list of Accepted Dental Remedies does not imply a recommendation. It means that the product and the methods by which it was marketed at the time of consideration were not found to be in violation of the Council's published rules. Accepted products are reconsidered periodically. The files of the Council contain information on many drugs and dental cosmetics. All information is available, upon request, and inquiries are welcomed. A postal card will bring a prompt reply.

DONALD A. WALLACE, *Secretary.*

COUNCIL ON DENTAL HEALTH

SUMMARY OF REPORTS RECEIVED FROM STATE COUNCILS ON DENTAL HEALTH

TEXAS

THE Council on Dental Health of the Texas State Dental Society was organized in accordance with the plan of the American Dental Association. The executive committee of the state council is appointed by the president and corresponds to the former council on mouth hygiene. Chairmen of district councils, appointed annually by the district presidents, appoint local councilors who assist in sponsoring and promoting local dental health activities.

The Texas Council on Dental Health has thus far concerned itself with the following problems: merit system status of the dental personnel in the state department of health; distribution of the *Dental Pictorial;* excuses for school children for dental appointments; high school Victory Corps program; mobile dental units and the itinerant dental service program, and state appropriations for the dental health program.

Owing to the unusual demand for dental service, all of the phases of the Victory Corps program were not carried out. Dentists throughout the state have cooperated by giving preference to high school students.

The state legislature has never appropriated funds for the dental health program. Federal funds and funds appropriated through the state maternal and child hygiene division are used to operate the dental division of the state department of health. It was recommended that the Texas State Dental Society, through the legislative committee, lend its organized effort toward securing adequate appropriations for the dental health program.

MASSACHUSETTS

The report of the high school Victory Corps dental program dated August 1, 1944, revealed that out of a total of 1,447 senior students enrolled in fifteen schools, 869, or 60 per cent, returned Victory Corps dental cards. Treatment was completed for 26 per cent of the students and treatment was started for 30 per cent, while no provision was made for 4 per cent of the students requiring treatment.

WISCONSIN

A meeting of the Brown County Dental Society with northeastern Wisconsin educators resulted in a decision to organize a more nearly complete school dental health program. The purpose of the meeting was to acquaint public authorities with the state dental society's plans for dentistry in the school health program. Health education will be integrated into most of the school subjects under the health program of the state department of public instruction, sponsored by the curriculum committee of the department.

Under the proposed program, less emphasis will be placed on physical education and more on a pattern of health behavior. Experimental centers have been selected throughout the state, consisting of cities and villages of various sizes. In each of these communities, dentists, physicians, nurses and boards of health will be asked to participate.

Dentists have been appointed by the oral hygiene committee of the Brown County Dental Society to visit high schools and examine seniors in connec-

tion with the Victory Corps program. The experiences gained through this project will be used as a basis for developing permanent dental health programs in all elementary and secondary schools in the postwar period.

ALLEN O. GRUEBBEL,
Executive Secretary.

COMMITTEE ON INTERNATIONAL RELATIONS

EMERGENCY EDUCATIONAL AND CULTURAL REBUILDING OF THE WAR-TORN UNITED NATIONS

STATEMENT OF POLICY BY U. S. DEPARTMENT OF STATE

WAR is destroying the educational and cultural organization of the countries occupied by the enemy. Universities, schools, libraries, museums and scientific laboratories have been wrecked or greatly damaged. Books and equipment have been stolen. Retreating Axis armies are likely to do still more damage.

Teachers, students and scientists have been singled out for special persecution. Many have been imprisoned, deported or killed, particularly those refusing to collaborate with the enemy. In fact, the enemy is deliberately depriving his victims of those tools of intellectual life without which their recovery is impossible.

Educational disorganization and economic and social distress are connected, one intensifying the other. Increasingly, the war-torn countries are very likely to suffer a decline of their standards of living and health to critically low levels. The whole people will suffer, but in a special degree the children. Such conditions unavoidably tend toward internal disorder and external difficulties, and may create new threats to the economic stability and political security of the world, on which, in fact, depend the well-being and peace of the American people.

The peoples who survive this ordeal will need help—in order to help themselves. They are facing enormous problems in rebuilding educational and cultural life, without essential facilities, and without adequate trained personnel. Plans for these tasks must be made now and the work undertaken as soon as possible.

Because of the unprecedented crisis which must be faced in this regard, the Department of State believes that the participation of the United States Government in an international program for the rebuilding of essential educational and cultural facilities of the war-torn countries in the period immediately following hostilities is an important service in the national interest and in the interest of international security and that steps looking to this participation should be taken.

In the department's study to date of the kind of program that would be practicable and desirable, certain conclusions have already become clear. It would be unwise for this Government to undertake to apply, much less impose, a foreign educational program or system in any liberated country, or to develop a program for the placement of American teachers in the schools of these countries, or for the preparation of textbooks in the United States for use in such schools.

In order to help the war-torn coun-

tries to help themselves in the rebuilding of essential educational and cultural facilities, the department proposes to collaborate for the time being with the Conference of Allied Ministers of Education in London, and to cooperate with the nations represented in this conference and the other United Nations and with the nations associated with the United Nations in the war in forming, as soon as practicable, a United Nations organization for educational and cultural reconstruction. It recognizes that a significant effort has already been made abroad and that useful work has been begun in the shaping of an emergency program to meet this need.

This program, it now appears, may consist of (1) assistance in the restocking of essential educational facilities, especially with books and scientific and other teaching aids, (2) assistance in the providing of opportunities for the training of carefully selected foreign students in American educational institutions, (3) assistance in reestablishing essential library facilities and (4) assistance in the recovery and the restoration to their rightful owners of educational, scientific, artistic and archival materials looted by the Axis countries.

In this program, as in all other activities in educational and related fields, the department will seek the advice and cooperation of other agencies and organizations, both governmental and private. It will attempt to operate in a manner equally advantageous to all the countries concerned. This reciprocal relationship is basic in any sound program of educational and cultural relations.

This statement concerning the participation of the United States in emergency restoration of essential educational and cultural facilities of the war-torn United Nations deals with only one of the important educational and cultural problems in the international field which are receiving active consideration. Also, of very great significance in the long-range furtherance of educational and cultural relations among nations, the department wishes to increasingly encourage democratic international cooperation in developing reciprocal and desirable educational and cultural relations among the nations and peoples of the world, especially looking toward the promotion of free and friendly intellectual intercourse among them in the interest of international peace and security.

No attempt is made here to deal with the important questions concerning the educational and cultural programs of the Axis countries.

DANIEL F. LYNCH, *Chairman,*
Committee on Foreign Relations.

HORACE WELLS CENTENARY CELEBRATION

THE program for the Horace Wells Centenary Celebration in Hartford, December 11, 1944, is taking shape rapidly under the guidance of the committee appointed to make the arrangements. The list of speakers for the day is a formidable one, but since arrangements have been made to have concurrent meetings during the morning in both the auditorium of the Hunt Memorial on Prospect St., and in the Avery Memorial nearby, the time schedule can be kept, while, at the same time, permitting delegates and guests a choice of speakers.

The morning session will start at 9 a.m. and continue with occasional recesses until the luncheon meeting at 1:30 p.m. Howard R. Raper, D.D.S., of Albuquerque, N. M., will speak on "Glimpses of the Man, Horace Wells." Dr. Raper has for several years collected data dealing with Wells' life and his contribution to science. Theodor Blum, D.D.S., M.D., of New York, N. Y., will speak on "History and Use of Local Anesthesia in Surgery." E. A. Rovenstine, M.D., professor of anesthesia, New York University College of Medicine, and president of the American Society of Anesthetists, will have as his topic "Nitrous Oxide, Highlights and Sidelights." Charles J. Wells, M.D., of Syracuse, N. Y., will speak on "Horace Wells, the Discoverer of Anesthesia." There will be three or four other speakers, including Prof. Arno B. Luckhardt, of Chicago, Ill. The committee plans to obtain an outstanding surgeon to speak on anesthesia and general surgery; and an anesthetist or surgeon from the Army or Navy to speak on anesthesia in the armed forces.

At the luncheon meeting, there will be short speeches by Gov. Raymond E. Baldwin; Sen. Francis Maloney; H. Gildersleeve Jarvis, president of the Connecticut Medical Society; Walter H. Scherer, Houston, Texas, President of the American Dental Association, and Arthur H. Merritt, Past President of the American Dental Association. The main address will be given by Thomas Parran, Surgeon General of the United States Public Health Service.

At some time as yet unscheduled, a plaque marking the site of the former home of Horace Wells will be unveiled.

Plans are under way for a nation-wide radio broadcast from the Hartford meeting.

The completed program and other information will be published as soon as possible.

The war and transportation difficulties have made impossible a nation-wide gathering in Hartford, Conn. The American Dental Association Committee therefore urges all dental schools and state, local and component societies to plan a Wells Centenary meeting in their respective localities for December 11, 1944, to commemorate this great event.

Essay and Program Committee:

ARTHUR H. MERRITT, D.D.S., A.D.A., *Chairman.*

HERBERT THOMS, M.D., Connecticut State Medical Society.

A. E. ROVENSTINE, M.D., President, American Society of Anesthetists.

SOCIALIZED DENTISTRY IN SWEDEN*

FOR some years before the present world war, we were cognizant of the changes taking place in the social structures of the United States and other countries. In an endeavor to learn the effect that these changes have had on the dental profession, I have spent the past year studying this question in Europe for the American Dental Association. Five months of my time was spent in Sweden, where socialized medicine has been in operation for many years and socialized dentistry was put into operation six years ago. This report will be confined to my investigations in Sweden, the country which is pioneering in the socialization field.

In my study in Sweden, I received the full cooperation of all state departments concerned, and was given access to records and accorded the privilege of visiting hospitals and clinics, with freedom to question employes and patients. Everything possible was done to enable me to secure a true picture of the functioning of socialized dentistry.

Many conditions influenced the passage of the *Folktandvard* law (people's dental care) and determined the provisions it contains. Some of these were: the character of the Swedish people; the financial status of the people; the tenets of the political party in control; the dental needs of the people and the number of dentists, and the previous socialization of medicine.

The people of Sweden are industrious, independent, democratic and realistic. They have achieved a very real concept of equality. Every man feels himself the equal of his neighbor, without regard to possessions. Respecting law and order, he will accept regimentation if convinced that it is for the public good. He has an intense personal pride which precludes the acceptance of charity. He reveres money and property far less than those titles evidencing superior knowledge and intellect. These traits of the Swedish national character reflect themselves especially in the monetary provisions of the *Folktandvard* law.

Sweden is not a wealthy country, the vast majority of its people being in the lower income bracket. The cold far north of the country, sparsely settled, affords mining and forestry employment. The warmer south is agrarian, comprised for the most part of small farms. The center of the country is given largely to industry, with resultant density of the population. The wealth is largely concentrated in the industrial areas and in the agrarian County of Skane in the south.

The Social Democratic party, depending largely on the strong labor and farm unions for its power, is in control of the political policy of Sweden. The policy of this party is decidedly socialistic and realistic, as is evidenced by a distinct trend toward cutting down the larger incomes. Political expediency has dictated the inclusion of adult dental care in the law, in order to facilitate its passage and minimize its cost.

The over-all condition of the people's teeth is poor. The incidence of caries varies greatly. In some counties, notably those in the industrial area, it is fairly good, while, in other counties, the prevalence of caries is appalling. Sweden has approximately 2,500 dentists to care for the dental needs of 6,500,000 people. The law of supply and demand makes it possible for these dentists to command high fees and have full appointment books. As a result, dental care is practically restricted to the well-to-do, dwelling, for the most part, in the industrial areas and cities.

The shortage of dentists is explained by the educational system. All higher education is controlled and directed by the state. Fees paid for courses vary greatly. Dental students pay the highest educational costs, whereas other students, particularly engineering, are favored by exceptionally low fees. There is only one dental school in Sweden, which is not only expensive, but also extremely difficult to enter. Only those students having the highest marks in their preliminary schools are admitted. The three teaching professors plan the policy and curriculum. Exclusive of the dental nurses' course, the student body numbers 300. The dental studies are carried on for four years. Examinations for advancement and graduation are conducted orally and in private.

*Report to the American Dental Association.

Graduation carries with it the license to practice dentistry.

The exclusiveness and expense of dental education has made dentistry in Sweden practically a closed corporation. (I have been informed that the dental school is the only institution showing a profit to the state.) The dearth of dentists, with resulting high fees, precluding dental care even for people of moderate means, greatly influenced the enactment of *Folktandvard*. Unfortunately, the law makes no provision for the building up of an adequate dental corps.

There is a wide schism between dentistry and medicine. Medical courses, given by several schools, are very thorough, requiring from seven to ten years for completion. Medicine has been socialized for more than a century and the service provided is without parallel. The state provides excellent facilities. Hospitals are roomy, lavishly equipped and adequately staffed with salaried doctors. All of the people, regardless of financial status, receive medical and surgical care at a nominal cost of 50 cents per day for inpatients and 25 cents per visit for outpatients. Wards contain four to six beds. Serious cases are cared for in private rooms. Socialized medicine in Sweden has made available to all the people the best in medical care.

Following is a résumé of Swedish dental service in one of the poorer counties, Halland, given me by the *Folktandvard* inspector of that county.

In 1870, a Danish dentist visited Halland occasionally, extracting and cutting off teeth, then taking an impression and returning to Denmark. In due time, the people received their dentures from him by mail. Many people did not have the money to pay for this service.

In 1880, the first Swedish dentist came to Halmstad, in Halland. Transportation was difficult and the village blacksmith did most of the extracting in the villages.

In 1918, when I graduated and went to Halmstad, there were fifteen dentists practicing in Halland. In those days, just after the close of the first world war, the people had money, prices of farm produce were high and people had their teeth taken care of. Dentists were busy and making plenty of money. Many students were impressed by this and studied dentistry. The hard times came and the farmer had no money for such luxuries as false teeth. It became a critical time for young dentists. The teeth of the people, especially women and children, were very bad. The Swedish Red Cross became interested. From America and England, the doctrine of focal infection became known. Many immigrants to the United States returned home and told of the dental treatment in America. The Red Cross and dental societies succeeded in starting dental clinics in the schools. In due course, the *Riksdag* (legislature) took notice and established dental treatment for the whole country.

In 1907, a group of dentists in Goteborg, moved by the condition of the teeth of the school children, endeavored to have the school authorities establish dental clinics. This effort was unsuccessful, but they did receive permission to treat several classes of children in their dental offices. The results were so spectacular that, in 1908, the City of Goteborg installed school clinics. At present, 21,500 children are cared for in these clinics in Goteborg. Children expecting to start school in September are required to register in May of the same year. At the time of their registration, their teeth are examined and any first permanent molars needing attention are cared for during June. In 1908, Stockholm inaugurated school clinics, where, at present, 30,000 children are cared for. Other cities have also established school clinics. These clinics are providing complete dental service (including orthodontia) of a very high order, and have done much to improve the dental conditions of the youth of the country. The smaller schools and those in rural districts, however, have not received the benefits of dental clinics. The excellent work done by the school clinics was influential in the inauguration of *Folktandvard*.

Folktandvard is now taking over the school clinics. This is causing some dissension, as the school day and year allowed for shorter working time than will *Folktandvard* clinics, while the pay will be substantially the same.

The Eastman Institute was established in Stockholm about ten years ago. The Institute registers children at 2 years and treats them until they reach the age of 15. It has pediatric, nose and throat and orthodontia departments. The services rendered by this laudable institution, made possible by the generosity of the late George Eastman, are excellent and a very valuable adjunct to the

service rendered by the school clinics in Stockholm.

Just after the turn of the century, the first movement was made to socialize dentistry. In 1904, an attempt was made to have the *Riksdag* (legislature) take action on this question, but it was unsuccessful. Similar unsuccessful attempts were made in 1913 and 1920. It was not until 1938 that the *Folktandvard* law was passed, to take effect in 1939.

(Sweden is divided into 25 *Lans,* similar to our counties and having their own county governments. The rate of exchange at present is 4.18 *Kronar* to the dollar. However, in normal times, the purchasing power of a *Krona* was equal approximately to the normal purchasing power of 50 cents here. All state employes in these abnormal times are given an increased cost of living bonus, ranging from 19.5 per cent in rural communities to 45 per cent in Stockholm. *Folktandvard* employes receive this bonus.)

June 3, 1938, Bill 358 and 359 relating to *Folktandvard* was passed by the *Riksdag.* These laws have since been amended by 351/1940, 1058/1940, 422/1942 and 955/1942.

358/1938 provides that:

1. State subsidy may be given to any *Lan* that arranges to give *Folktandvard* to children and adults according to certain specifications.

2. Each *Lan* shall appoint a *Folktandvard* committee in its *Landsting* (county government). They shall also appoint a *Folktandvard* inspector, preferably the chief of the central dental clinic (explained later), who shall sit with the *Landsting* and shall be heard, but shall not be entitled to a vote. His opinions, however, must be noted on the minutes of the meeting.

3. Each *Lan* shall be divided into districts according to plans approved by the *Medicinalstyrelsen* (surgeon general). Each district shall have at least one permanent clinic. At each clinic, at least one dentist and one registered dental nurse shall be employed. In districts that are very large, or that have transportation difficulties, there may be one or more ambulatory clinics.

4. As a complement to the district clinics, there shall be in each *Lan* a central dental clinic, attached preferably to the *Lan* hospital. Here, complicated cases referred from the district clinics and inpatients shall be treated. The chief of the central dental clinic must have special qualifications in surgery, prosthesis, orthodontia and children's dentistry. He shall also be the *Folktandvard* inspector of the *Lan* and make regular inspections and reports, according to *Medicinalstyrelsen* directives.

5. Each district dentist shall work 1,900 hours per annum, 1,000 of these hours to be spent treating children. Children are to be registered and treated from age 3 to age 15 and must be seen annually. Each child will pay 5 *Kronar* per year. In families where there is more than one child registered in *Folktandvard,* the fee for the second child will be 3 *Kronar,* and for a third child, 2 *Kronar,* any additional children in one family receiving treatment gratis. A child not registered with a clinic may receive treatment at 75 per cent of the adult rate. Adults are to be treated and charged fees according to the schedule issued by the *Medicinalstyrelsen.*

6. Dentists in *Folktandvard* shall be licensed practitioners. Dental nurses shall be graduates of the Swedish dental school or of the Eastman Institute.

7. District dentists shall have a salary of 7,200 *Kronar* per year and 10 per cent of the fees collected by him. For each three years of service, he shall receive an increase of 500 *Kronar* per year, up to and including three such increases. District dental nurses shall receive 3,000 *Kronar* per year and for each three years service shall receive an increase of 100 *Kronar* per year, up to and including three such increases. District dentists and nurses shall receive thirty days' annual leave and may receive thirty days' sick leave. They shall also be enrolled in the state pension fund without cost.

8. State subsidy is paid partly at the beginning as an equipment aid and partly each year as an aid toward salaries to the district dentist and the district nurse. State subsidy for equipment shall be one half the cost of such equipment up to 3,000 *Kronar* for a clinic employing one operator, with an additional subsidy of 1,300 *Kronar* for the equipment for each additional operator. For each ambulatory clinic, the subsidy shall be not more than 1,000 *Kronar.* A state subsidy for salaries shall be 3,000 *Kronar* per annum for each dentist and 1,000 *Kronar*

per annum for each dental nurse. Increases for length of service will be paid by the state. No dentist employed in *Folktandvard* may have a private practice.

Bill 359/1938 (Rates for Adults): The rates given in the accompanying table are not valid for children between 3 and 15 years of age who have applied for regular enrollment in *Folktandvard,* nor for complicated cases referred to the central dental clinic.

CLINIC RATES

	Kronar
Consultation	1
X-ray film (single)	3
X-ray film (full mouth)	15
Prophylaxis, including gum treatment	1-5
Root amputation or cyst removal	20
Treatments	1
Extraction:	
Without procaine, one tooth	2
Each additional tooth, same sitting	1
With procaine, one tooth	3
Each additional tooth, same sitting	1
Maximum charge, one jaw	15
Maximum charge, both jaws	25
Difficult cases (impactions)	15
Fillings:	
Amalgam	4-10
Silicate	5
Gold inlays	20-30
Temporary fillings	1
Root canal (treatment and filling)	11
Prosthesis:	
Davis crown gold base	25
Richmond crown	45
Cast gold crown	40-50
Rubber denture, first tooth	15
Each additional tooth	2
Full upper or lower	40
Full upper and lower	70
Acrylic, full upper or lower	70
Full upper and lower	120
Fitting of denture	5

Youths between 15 and 19 years of age receive 25 per cent reduction. For treatment not included in the schedule of fees, the fee shall be 7 *Kronar* per hour. Patients must pay for work done at the time of each visit. The dentist is responsible for the records and payments of his patients.

Bill 360/1938 (Community Subsidy): Municipalities and communities in a *Lan* shall pay the fees for the children of indigent parents. They shall also pay the fees for work done for indigent adults. One half of these fees will be returned as a subsidy by the state. The state will not pay for any precious metals or porcelain used for these patients. Any fees so paid shall not be listed as poor relief or so considered.

Bill 955/1942: (Induction of Recent Graduates of Dental School): All graduates of the dental school will work one year in *Folktandvard,* before they can be licensed to practice dentistry privately.

Such is a summary of the laws governing *Folktandvard.* Salaries to be paid are the minimum ones allowed. Each *Lan* makes its own arrangements as to salaries. It is common practice in nearly all *Lans* to pay the base pay of 7,200 *Kronar* and 10 per cent commission on the first 8,000 *Kronar* collected in fees, 30 per cent commission being paid on any additional fees collected. Individuals have been known to contract for 40 per cent of their fees above 8,000 *Kronar.*

The average earning of a dentist in *Folktandvard* is about 12,000 *Kronar* per year. In a few *Lans,* the average is higher, being 14,000 to 15,000 *Kronar* per year. These figures are not very informative unless one knows the country. A professional man in Sweden must maintain a certain standard of living. Call it, "Keeping up with the Joneses," if you will. A dentist, to maintain his social position, requires at least 15,000 *Kronar* per year. In contrast to the earnings of a district dentist in *Folktandvard,* a recent graduate in dentistry who becomes the assistant to an established practitioner can earn 20,000 *Kronar* per year.

The clinics provided by the state are ideal. In most cases, where the population warrants it, especially designed buildings are erected. All clinics are complete in every detail and lavishly equipped. No private practitioner could ask for a better office than is provided by *Folktandvard.*

Since its inauguration in 1939, *Folktandvard* has expanded slowly. Originally designed to employ 1,000 dentists, it has in its employ at present about 250. Of these, at least 10 per cent are refugees, who cannot be considered permanent employes. The graduating class of 1943 is now serving in *Folktandvard.* It is problematic how many of this group will continue to serve when the year of compulsory service in *Folktandvard* is finished.

The War Defense Program has undoubtedly interfered with the recruiting of dentists for *Folktandvard.* Dentists are called to

serve for a period each year in the military establishment.

As the caries incidence varies widely in different *Lans,* so the picture varies. I will cite the cases of two *Lans* in illustration.

The *Lan* of Sörmland, which lies in the central industrial area and is one of the more prosperous *Lans,* began its *Folktandvard* organization in 1939. The children in this *Lan* had already had the benefit of school dental clinics. The incidence of caries here is relatively low. The *Lan* has thirteen district clinics, augmented by a central clinic at the hospital in Eskilstuna. In 1943, there were thirty-three dentists employed who cared for 25,000 children and 16,000 adults, drawn from all walks of life. The number of children cared for represent 92 per cent of those in the urban areas and 80 per cent of those in suburban areas. The average earnings of the dentists in these districts was about 15,000 *Kronar.* Both the district dentists and the private practitioners in this *Lan* are contented and busy.

Receipts from *Folktandvard* during 1943 were in *Kronar:* From children, 80,947.00; From adults, 464,907.71; State subsidy, 113,817.43; Community, 39.443.40; Other income, 15,649.09; *Lan* subsidy, 112,029.45; Total, 826,694.08.

To this must be added an inventory of supplies, etc., at the end of the year, amounting to 120,000 *Kronar.* I have been informed that some *Lans* actually show a profit on their *Folktandvard.* This may be one of these *Lans.*

In the clinics of this *Lan,* I examined the mouths of 100 children who were making their annual visit. I was able to find only four cavities, one of which was a recurrent decay. I was impressed by the excellence of the work previously done, every filling having been carved and polished. This standard of excellence I found in the *Folktandvard* clinics generally. Sörmland has all the dentists that the law allows: i.e., one dentist to each 1,000 children in the *Lan.*

In Halland *Lan,* which lies on the south west coast of Sweden, there is very little industry. This is a farming, fishing and forestry region. The population is widely scattered and, on the whole, poor. Here, the incidence of caries is very high. Except for a few cities of moderate size, there has been no school dental care. It has been difficult to secure dentists to work in this *Lan,* as their earnings are in keeping with the general poverty of the section. In one of the small clinics located in the forest belt, I examined twenty-five children who had had their mouths completely taken care of one year previously. The condition of their teeth was extremely bad. In every mouth, decay was rampant. At least twenty of the children were anemic, a like number had septic tonsils and all appeared to be undernourished. There seemed to be no liaison between medicine and dentistry. The dentists in these outlying districts deserve the highest commendation for their perseverance in and conscientious devotion to the care of these children. It is in a *Lan* such as this that the inadequacy of the present *Folktandvard* establishment is revealed.

The shortage of dentists has done much to hamper the functioning of *Folktandvard.* It is noticeable, however, that what dentists there are in the service are being utilized to a large extent in locations that have had access to some dentist in the past. These places are in a better financial condition to establish clinics.

It is natural for the dentist, because of the commission basis of pay, to seek out locations where the most money can be earned. *Lans* such as Sörmland and Skane, where there is money, have a full complement of operators. *Lans* such as Halland, where the financial condition is poor, cannot compete with the richer *Lans* for dentists. Peculiarly, although the law allows the use of ambulatory clinics, I saw none in operation. This, in spite of the fact that, in the sparsely settled areas, it would mean reaching those most in need of care.

In some district clinics that employ several dentists, there has been a departure from the regular procedure. Some operators treat children only and are given a base pay of 9,000 *Kronar.* Their opposite number treats adults exclusively, except for 100 hours per year. Thus, between two operators, 2,000 hours of children's dental care is given.

The registered dental nurses have completed a two-year course. They make no examinations and give no prophylaxis, their training being confined to chair assistance, sterilization and clerical work.

In visiting the various clinics, I asked many questions of the staffs. The chief criticism was that "the pay is too small." The majority of dentists in *Folktandvard* are primarily interested in improving the dental condition of the people. Occasionally, I met a dentist who was trying to put the state in the dental business. These would complain that they need more time for treating adults. I found one chief of a clinic who was trying to circumvent the regulations calling for assignment of one dentist for each 1,000 children. She complained bitterly because she could not get two additional dentists to care for adults, who, she said, wanted to leave the private practitioners and have their work done in the *Folktandvard* clinic.

Following are some of the responses to my questions concerning the operation of *Folktandvard:*

In view of the limited number of dentists available, would it not have been better to start Folktandvard as an extension of the school dental clinics, caring for the children in outlying districts where the conditions are so bad?

There is no doubt that children should be cared for first. We did not anticipate the difficulty that we are having in securing dentists. In order to get the necessary appropriation without too much opposition, we considered it wisest to do something for the adults. They pay the taxes. (Answered by state official.)

Why was the salary of dentists placed at 7,200 Kronar, whereas a private practitioner can earn so much more?

The state employs various professional people: lawyers, engineers, etc. These men have all spent a like number of years being educated. The other professional men employed by the state are paid on the same level as we are paying the dentists. (State official.)

Why was no attempt made to restrict adult dental care to people of low income, and why are adult fees more than cost?

Our people will not accept charity. They insist on paying a fair fee. Provision is made to take care of indigent people and expectant mothers before the general adult public. However, *Folktandvard* is a state institution and all citizens are entitled to its opportunities and benefits. No question of financial status can be raised. The fees paid by adults in excess of costs help to defray the expense of caring for children, thus lightening the burden for the taxpayer. (State official.)

In my *Lan,* I have a long waiting list of adult patients. The poor, who cannot afford to pay, are placed at the head of the list. My dentists cannot develop so large an income as in some other *Lans,* but I expect to get them an increase in pay. *(Folktandvard* inspector.)

We take the people as they apply, all being treated equally. If my dentists can make acrylic plates, it increases their income and they are better satisfied. (Chief of dental clinic of another *Lan.*)

The private dentists in this town get very little prosthetic work to do any more. The *Folktandvard* clinic gets that class of work because their fees are lower than ours. We get the fillings to do. (Private dentist in the same town.)

All dentists of the class of 1943,* who are forced by law to work one year in *Folktandvard,* were asked the following question:

How do you feel about Folktandvard? Do you intend staying in it?

The answers to the foregoing questions were practically all identical.

I think the law is unfair. We were all in debt when we graduated. I owed my parents 20,000 *Kronar.* This must be paid back. In *Folktandvard,* it will take many years to do this, as I can save very little out of what I get. I married a classmate. He has to serve his year of military service in addition to his year in *Folktandvard.* When he completes these two years of service, he will go into private practice. This means he has lost two years. I shall stay on until he is established, then I will go with him. We cannot afford to stay. The work is interesting and needs to be done. If the pay were better, we would stay in it. (District dentist.)

The work is interesting and essential. I enjoy helping these people; but, next month, I must go into the Army for a year. Then I must come back to finish my year here. I wish to get married and, before I do, I wish to be out of debt.† I cannot get out of debt on what I make here. As soon as I can, I shall go into private practice in order to make some money. (District dentist.)

Since dental education is expensive, leaving students deeply in debt, why does not the state make some provision to assist the students who are forced to work in Folktandvard?

At present, we are endeavoring to make

*The class of 1943 was about equally divided between male and female, as is the profession generally.

†Virtually every student is meticulous about returning to those who educated him the money borrowed for his education.

some arrangement, along this line. Some such provision is indicated and will be worked out in connection with the intended expansion of our dental education facilities. (State official.)

In discussing *Folktandvard* with private practitioners, I found them to be interested in the subject and not antagonistic. Some were disappointed that the problem had not been handled more scientifically and with more benefit to the people in outlying districts. I quote some of their comments:

"I think they *(Folktandvard)* have a great opportunity to do something not only for the people, but also for dentistry. If they would go into the places where the people are sorely in need of dental care and do some research on the effect of nutrition and hygiene on caries, all would be benefited."

"*Folktandvard* is a fine idea, but when you ask a man with a practice to give it up and accept a lower income, it is a trifle foolish. With responsibilities that such a man has taken on and the years required to reach a good income, it cannot be done. What would become of our patients? Would they not suffer for lack of care? No, the answer is: More dentists. *Folktandvard* should have known they would need more men before they began."

A distinguished sociologist commented: "Dentistry is a public health service; it should be a profession and not a business. The dentists of this country have been making too much money and neglecting their duty as a profession. It is the state's duty to care for its people; no one should profit from ill health."

One state official with whom I spoke (I am happy to say that he was the only one, among the many state officials interviewed) seemed to think that the main idea behind *Folktandvard* was to reduce the income of dentists. He was convinced that if all lived in poverty, none would be unhappy.

CONCLUSION

I shall not draw any conclusions. I have endeavored to be reportorial rather than editorial. *Folktandvard* is growing and I am told that money will be appropriated for research, that a greater degree of cooperation with the medical profession is contemplated and that an investigation is now under way for enlarging the present dental school and creating another one.

In my travels over Sweden, I was greatly impressed by the desire of the dentists to stay abreast of any advancement in the profession. Many of them are occupied with research. They are studious and skilful, a credit to the dental profession.

Owing to war conditions, there is a shortage of dental supplies, especially those manufactured in the United States, which are greatly favored. Sweden is manufacturing its own instruments, their surgical and diamond instruments being the finest I have ever seen.

In closing this report, I wish to thank *Statsrod Möller* (the social minister), Dr. A. Hoyer, director, *Medicinalstyrelsen,* Dr. A. Maunsbach, director, *Folktandvard* and his field assistants for their courtesy and assistance. Without their cooperation, my study could not have been made.

NILS HENRY LARSON, D.D.S.

COUNCIL ON DENTAL EDUCATION

GRADUATES OF DENTAL SCHOOLS IN THE UNITED STATES FOR THE YEAR ENDING JUNE 30, 1944

School	Predental College Training						Total	Per Cent with Degree	No. Graduating in Combined Courses
	Less Than Two Years	Two Years	Three Years	Four Years Without Degree	Bachelor's Degree	Other Degree			
Physicians & Surgeons		25	5	1	5	1	37	16	8
California		45	10	2	11	0	68	16	68
S. California		60	11	3	9	1	84	12	26
Georgetown		22	9	2	13	0	46	28	0
Howard		8	5	0	12	0	25	48	0
Atlanta		68	14	2	18	0	102	18	0
Loyola, Chicago		58	12	0	8	0	78	10	0
Northwestern		41	8	0	24	0	73	33	0
Illinois		29	8	2	13	0	52	25	34
Indiana		42	16	6	13	0	77	17	8
Iowa		34	15	0	12	0	61	20	18
Louisville		15	14	1	9	0	39	23	0
Loyola, New Orleans		34	10	1	15	0	60	25	0
Maryland		46	10	0	18	0	74	24	0
Harvard		16	6	0	15	1	38	42	0
Tufts		35	11	1	26	1	74	36	0
Detroit		23	4	1	7	0	35	20	0
Michigan		37	20	0	28	3	88	35	7
Minnesota		37	14	2	1	3	57	7	0
Kansas City	1	92	15	2	20	0	130	15	0
St. Louis		27	11	6	8	4	56	21	0
Washington		8	2	0	5	1	16	37	0
Creighton		13	4	1	1	1	20	10	3
Nebraska		10	2	0	2	0	14	14	12
Columbia		0	12	0	39	0	51	76	2
New York		9	22	2	103	4	140	76	17
Buffalo		22	12	1	11	0	46	24	0
Ohio		26	14	1	14	0	55	25	0
Western Reserve		17	6	1	8	0	32	25	12
North Pacific		19	7	2	8	4	40	30	9
Temple		123	35	6	44	1	209	22	0
Pennsylvania		36	22	1	32	3	94	37	0
Pittsburgh		56	24	4	69	3	156	46	20
Meharry		2	1	0	6	0	9	67	0
Tennessee		9	6	2	9	0	26	35	0
Baylor		21	9	1	5	0	36	14	0
Texas		23	3	1	4	0	31	13	0
Virginia		13	7	1	5	0	26	19	0
Marquette		61	19	2	28	5	115	29	1
Totals	1	1,262	435	58	678	36	2,470	29	245

CORRESPONDENCE

"COMPLETE DENTURES IN TWENTY-FOUR HOURS"

To the Editor:

The article "Complete Dentures in Twenty-Four Hours," published in THE JOURNAL, September 1, 1944, has caused a great deal of unfavorable comment from various sections of the country.

This article, written by a Public Relations Officer of the Fourth Service Command, was not approved or seen prior to publication, by Col. Tyler J. Walker, chief of the Dental Service at Oliver General Hospital, by the Camp Dental Surgeon at Camp Gordon or by Col. Robert Tobias, Fourth Service Command Dental Surgeon. Furthermore, the article was never approved or seen in the Dental Division, Surgeon General's Office.

The pictures in the article were taken by the Signal Corps Photographic Laboratory, and the Public Relations Officer furnished the writer of the article with some general information, including *one case* wherein the dental service was called upon to complete a particular denture in a limited time. The title was selected on the basis of this one case, which implies that many dentures are made in twenty-four hours. The fact is that it requires about ten days to two weeks to complete the average denture.

The Army Dental Corps does not recommend, approve or practice "Complete Dentures in Twenty-Four Hours." There are very exceptional cases when every effort is extended to complete a particular denture in a limited amount of time for a key individual. It is impractical, inefficient and out of the question to produce the average denture in less than one week's time, and many require two weeks.

I do not criticize you in any way, since you had a War Department Bureau of Public Relations' stamp of approval on the manuscript, and you assumed that it had the full approval of the Army Dental Corps. Unfortunately, this office was not asked to review this material. I hope that you will refer to this unfortunate incident and clarify the position of the Army Dental Corps in your next issue.

It is further desired that no article regarding the Army Dental Corps be published in THE JOURNAL unless it has the approval of the Surgeon General's Office. I am sure you will give this office your full cooperation in this respect.

Yours sincerely,

R. H. MILLS,
Major General, U. S. Army,
Director, Dental Division.

"SIALADENITIS AND SIALODOCHITIS DIAGNOSIS AND MANAGEMENT"

To the Editor:

In the August 1, 1944 issue of THE JOURNAL, an article appeared entitled "Sialadenitis and Sialodochitis Diagnosis and Management," by George D. Wolf, M. D., and David Soloway, M. D.

It is not my intention to pass upon the merits of an article, especially when it is authenticated by the acceptance of a responsible publication. These authors, however, unfortunately make the dogmatic statement that "when the stone is in the body of the gland, the extirpation of the gland is the only means of effecting a cure." (P. 1035.)

I have personally treated scores of cases, as have others engaged in the field of oral surgery, in which the calculus was lodged in the substance of the gland. The parenchyma of by far the major part of the gland remains unaffected and the stones were removed in all instances intra-orally while the gland was left *in situ*. Its function continues uninterruptedly and permanently. On the other hand, I have in several cases, seen, besides the external scarring, other unfortunate and permanent complications of removal of the gland.

I do not desire to take exception to modes of treatment or procedure at the hands of other professional men since many considerations enter into the procedures of surgical practice. The point I wish to establish, however, is that these cases can be and are being frequently cured without removing the gland. When the gland is removed unnecessarily, the patient is deprived of the function of an essential organ.

ADOLPH BERGER, M.D.,
654 Madison Avenue,
New York 21, N. Y.

OBITUARY

FREDERICK B. MOOREHEAD, B.S., D.D.S., M.S., M.D.
(1875-1944)

FREDERICK BROWN MOOREHEAD, eminent Chicago oral surgeon and dental educator, died August 29, 1944, after a prolonged illness. Because of his keen interest in the dental profession and his enthusiastic support of the finest elements in dentistry, Dr. Moorehead was a great force in elevating the dental profession. His leadership will be missed, but his ideas and ideals will be put in practice by many hundreds of his students.

Dr. Moorehead was born in Mineral Point, Wis., October 14, 1875. He was graduated from the Chicago College of Dental Surgery in 1899, and received the degree of bachelor of science from the University of Chicago in 1904, the degree of master of science from the University of Michigan in 1906 and the M.D. degree from Rush Medical College in 1906.

Dr. Moorehead's chief contributions were in the field of dental education, for he served as dean of the University of Illinois, College of Dentistry from 1913 to 1926 and as professor and head of the department of oral and plastic surgery from 1913 until his death. During all these years, he insisted, constantly, on the highest educational standards in the dental college. He was senior author of the textbook "Pathology of the Mouth," and contributed many articles to the current literature. He was also active in Rush Medical College and served on the staff of the Presbyterian Hospital, Children's Memorial Hospital and the Home for Destitute and Crippled Children.

All this he did in addition to maintaining a busy practice in oral surgery.

Dr. Moorehead joined the American Dental Association in 1905, and maintained his membership continuously until his death. He acted as Delegate or Alternate to the American Dental Association three times. He was a member of the American Medical Association, the Chicago Dental Society (president, 1915), the Institute of Medicine, the Chicago Pathological Society, the American Association of Oral and Plastic Surgeons (president, 1926-27), Delta Sigma Delta and Nu Sigma Nu. In 1901, he married Marguerite Hirst. They had two children, Chester and Dorothy. He was married a second time (1929), to Margery Maxwell, of the Metropolitan Opera.

FREDERICK BROWN MOOREHEAD
(1875-1944)

Always a forceful speaker, Dr. Moore-

head enlivened many meetings with his discussions and comments. His influence played an important part in elevation of the standards of dental education, and dentistry owes him a great debt.

G. WALTER DITTMAR.

DEATHS

ABBEY, WILLIS F., Hartford, Conn.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1913; died August 20.

ABBOTT, CHARLES F., Martinsville, Ind.; died August 18; aged 38.

ABBOTT, RUFUS, Woodward, Okla.; died May 19; aged 85.

ALBRECHT, CHARLES H., Waukegan, Ill.; Northwestern University Dental School; died August 30; aged 80.

ALZENO, GUERNEY E., Stockton, Ill.; Northwestern University Dental School, 1915; died Sept. 4; aged 59.

ARDE, WALKER R., Philadelphia, Pa.; Pennsylvania College of Dental Surgery, 1900; died August 23; aged 70.

ARROWSMITH, JOSEPH B., Erie, Pa.; Medico-Chirurgical College of Philadelphia, 1904; died September 6.

BARRETT, WALTER O., Jamestown, N. Y.; University of Buffalo, School of Dentistry, 1921; died September 10; aged 47.

BERG, PAUL I., Rockford, Ill.; died August 24; aged 45.

BERRY, J. O., Provo, Utah; Chicago College of Dental Surgery, Dental Department of Loyola University, 1920; died August 22; aged 51.

BITTNER, U. J., Redmond, Ore.; North Pacific College of Oregon, School of Dentistry, 1911; died July 31; aged 57.

BLYMEYER, GEORGE W., Parkesburg, Pa.; Pennsylvania College of Dental Surgery, 1903; died August 23; aged 66.

BOND, JOHN L., Earl Park, Ind.; Northwestern University Dental School, 1902; died August 14; aged 65.

BOOKER, FRANK D., Englewood, Calif. (formerly of Rochester, Minn.); Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania, 1900; died September 9; aged 69.

BROCK, HENRY CLAY, North Platte, Nebr.; United States Dental College, Chicago, 1898; died June 27; aged 67.

BROWN, GODWIN M., Newton, Mass.; Temple University School of Dentistry, 1894; died September 22.

BURLING, PHILIP R., Oak Park, Ill.; Northwestern University Dental School, 1940; killed in performance of duty in the Himalayas April 28. Dr. Burling was a captain in the U. S. Army Air Corps.

CAMPBELL, JAMES FREDERICK, Hickory, N. C.; Atlanta-Southern Dental College, 1918; died August 12; aged 48.

COLLINS, F. B., Springdale, Ark., died August 20; aged 70.

COX, JACKSON, Huntington Park, Calif.; died August 22; aged 35.

CRAWFORD, L. H., Crane, Texas; died May 29; aged 67.

CROCKER, George K., Cincinnati, Ohio; died September 6; aged 61.

CROMIE, WILLIAM A., Beaver, Pa.; University of Pittsburgh, School of Dentistry, 1926; died August 19; aged 57.

CURTIS, S. W., Tipton, Ind.; Indiana University School of Dentistry, 1889; died August 30; aged 78.

DAY, JUDSON L., Clinton Falls, Minn.; University of Minnesota, College of Dentistry, 1903; died September 14; aged 67.

DAYTON, M. F., Seymour, Mo.; St. Louis University College of Dentistry, 1925; died August 17; aged 44.

DEALE, J. WARREN, Greenport, N. Y.; Pennsylvania College of Dental Surgery, 1909; died September 16; aged 59.

DONALDSON, M. R., Tiptonville, Tenn.; Vanderbilt University, School of Dentistry, 1907; died August 24; aged 58.

EASTMAN, WILLIAM C., Boston, Mass.; died September 14; aged 56.

ECHTERNACH, JOSEPH P., Philadelphia, Pa.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania, 1903; died August 22.

FOUTS, M. H., Mulberry, Fla.; Southern Dental College, Atlanta, 1912; died August 11; aged 58.

FOWLER, SHERMAN M., Muskegon, Mich.; University of Michigan, College of Dental Surgery, 1888; died September 21; aged 79.

FULLER, ALFRED D., St. Louis, Mo.; died September 6; aged 70.

GIBBONS, ROBERT E., Laurel, Md.; Baltimore College of Dental Surgery, 1900; died August 19; aged 73.

GILMAN, GEORGE W., Irvington, N. J.; New York College of Dentistry, 1916; died September 8.

GOUSE, M. W., Orangeville, Ill.; Northwestern University Dental School, 1904; died August 20; aged 62.

GRAFINGER, STEPHAN C., Cleveland, Ohio; died August 24; aged 44.

HANSEN, F. N., Columbus, Wis., died August 31; aged 67.

HANSON, J. A., Faribault, Minn.; University of Minnesota, College of Dentistry, 1914; died August 19; aged 56.

HAPPEL, E. P., Luxemburg, Wis.; Marquette University, School of Dentistry, 1912; died September 1; aged 61.

HARDING, H. G., Lewisville, N. C.; Atlanta-Southern Dental College, 1919; died August 28; aged 50.

HARRISON, JOSEPH LEE, Atlanta, Ga.; Atlanta Dental College, 1909; died August 25; aged 66.

HART, FRED E., Los Angeles, Calif.; State University of Iowa, College of Dentistry, 1895; died August 20.

HUFFMAN, HARVEY CARR, Dayton, Ohio; Ohio College of Dental Surgery, Dental Department of the University of Cincinnati, 1905; died September 3; aged 59.

HUFSTEDLER, P. A., Union City, Tenn.; University of Tennessee, College of Dentistry, 1918; died September 1; aged 55.

HUGGINS, JOSEPH, Downingtown, Pa.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania, 1897; died September 22.

KELLY, WILLIAM J., Wakefield, Mass.; Tufts College Dental School, 1897; died August 11; aged 73.

KILLINGSWORTH, SAMUEL F., Columbia, S. C.; Atlanta Dental College, 1895; died September 5; aged 76.

KING, HORACE W., Elmhurst, N. Y.; New York College of Dentistry, 1904; died September 20; aged 64.

KIRK, MILTON W., Seattle, Wash.; died August 24; aged 61.

KRAJEWSKI, THADDEUS, Middletown, Mass.; killed in action in France recently. Dr. Krajewski was a captain in the Army Dental Corps.

LAWRENCE, JAMES W., Langley, Wash.; Chicago College of Dental Surgery, Dental Department of Loyola University; died September 10; aged 65.

LEHMAN, FRANCIS E., New York, N. Y.; New York College of Dentistry, 1924; died September 16; aged 42.

LESURE, WILLIAM A., Boston, Mass.; Tufts College Dental School, 1891; died August 14; aged 75.

LIBBY, ARTHUR A., Boston, Mass.; Harvard University Dental School, 1899; died September 8; aged 68.

LUCKEY, CHARLES MOULTON, Plainfield, N. J.; New York College of Dentistry, 1891; died September 5; aged 75.

MCCARTHY, MARTIN, Watertown, S. D.; died August 26; aged 69.

MCDOUGALL, KENNETH, Stamford, Conn.; Pennsylvania College of Dental Surgery, Philadelphia, 1889; died August 14; aged 78.

MARTIN, ALBERT R., Los Angeles, Calif.; University of Southern California, College of Dentistry, 1919; died August 21; aged 47.

MERRITT, CLARK M., Lansing, Mich.; University of Michigan, College of Dental Surgery, 1908; died September 21; aged 71.

MONTGOMERY, JAMES C., Elizabethtown, Ky.; died May 27; aged 75.

MUNSON, CHARLES B., Arlington, Va.; Columbian University, Dental Department, Washington, D. C., 1889; died August 27; aged 75.

ORAM, WARREN W., Eagle Bend, Minn.; University of Minnesota, College of Dentistry, 1911; died September 1; aged 55.

PAGE, CHARLES, Chicago, Ill.; died August 29; aged 80.

PURVIS, S. P., Salisbury, N. C.; Baltimore College of Dental Surgery, 1907; died August 24; aged 67.

QUITER, R. J., Elmhurst, Ill.; Northwestern University Dental School, 1923; died August 30; aged 44.

RAZEY, J. HADLEY, Ellicottville, N. Y.; died September 1; aged 62.

REYNOLDS, FRANCIS B., South Amenia, N. Y.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania, 1901; died August 25; aged 68.

RUGGLES, STEWART DANA, Portsmouth, Ohio; Northwestern University Dental School, 1896; died September 6; aged 69.

SCHIFF, DAVID C., Peru, Ind.; Northwestern University Dental School, 1900; died June 19.

SMITH, SIDNEY, Paducah, Ky.; University of of Tennessee, College of Dentistry, 1902; died September 16; aged 67.

SPITZ, O. I., Chicago, Ill.; Chicago College of Dental Surgery, 1897; died August 27.

STEELE, GEORGE H., Caro, Mich.; Chicago College of Dental Surgery, Dental Department of Loyola University, 1904; died August 30; aged 63.

STOFFER, JOHN E., Lansing, Mich.; University of Michigan, College of Dental Surgery, 1900; died July 28; aged 74.

TATE, WILL I., Springfield, Ohio; Ohio College of Dental Surgery, Dental Department, University of Cincinnati, 1903; died August 3; aged 62.

TAYLOR, JOHN W., Covington, Ky.; Ohio College of Dental Surgery, Dental Department of the University of Cincinnati, 1897; died July 26; aged 87.

TOMMERSON, L. N., Hettinger, N. D.; University of Illinois, College of Dentistry, 1914; died July 31; aged 51.

TOWNSEND, ALMOND FRANCIS, Worcester, Mass.; Tufts College Dental School, 1881; died August 29.

TROTHAN, CHARLES W., Detroit, Mich.; Temple University School of Dentistry, 1904; died September 8; aged 64.

TURNER, CHARLES A., Statesville, N. C.; died September 15; aged 77.

VALENTINER, HAROLD OLAV, Miami Beach, Fla. (formerly of Cincinnati, Ohio); Cincinnati College of Dental Surgery, 1902; died in August; aged 64.

VANN, GEORGE S., Gadsden, Ala.; Vanderbilt University, School of Dentistry, 1889; died August 12.

VAN WINKEL, WILLIAM D., Port Chicago, Calif.; Marion-Sims Dental College, St. Louis, 1903; died June 30; aged 76.

VOEGE, H. O., Alton, Iowa; State University of Iowa, College of Dentistry, 1919; died August 15; aged 45.

NEWS

AMALGAMATION OF CONSTITUENT SOCIETIES

THE amalgamation of the constituent societies of the Allied Dental Council and the First and Second District Dental Societies is an accomplished fact. Almost 1,000 dentists in greater New York have become new members of the two district societies, the Dental Society of the State of New York and the American Dental Association.

Negotiations for amalgamation have been in progress for the past ten years. Inability to solve many knotty problems resulted in several impasses. However, the proponents of amalgamation persisted, obstacles were overcome and the wishes of a large number of the profession in New York City to have a united front in dentistry became a reality.

The First District Dental Society, which embraces Manhattan and Bronx Counties, had a total membership of approximately 3,700 October 1, 1944. The Second District Dental Society, which includes the boroughs of Kings, Queens, Richmond, Nassau and Suffolk, had a total membership of approximately 2,800 October 1, 1944.

Now that negotiations and the legal procedures have been settled, there is a fine prevailing spirit of cordiality and cooperation which augurs well for dentistry in Greater New York.

CURRENT LITERATURE

Secondary Pellagra. IV. Diseases of the Alimentary Canal (Mouth and Throat)

W. B. Bean, T. D. Spies and M. A. Blankenhorn

Secondary pellagra is that form of the disease secondary to some other disease process. Tooth extraction, dental infection, osteomyelitis and fracture and tumors of the jaws, by necessitating change from a balanced meat and vegetable diet to one of cereals, mush and liquids, appear to be important factors. Lesions such as those produced in Vincent's infection and thrush are definitely related to pellagra, but whether they contribute to pellagra or result from it is not known. Tonsillitis, diphtheria, scarlet fever and streptococcal pharyngeal infections are ordinarily not related to pellagra. However, severe throat infections in poorly nourished persons undoubtedly tend to further upset the nutritional balance of the vitamin B complex.—*Medicine*, 23: 1, 1944.

H. B. McCauley.

Diagnosis, Classification and Treatment of Tumors of the Salivary Glands

R. T. Payne, M. C. Tod and M. Lederman

Tumors of the salivary glands constitute from 1 to 2 per cent of large series of tumors. They may originate in or adjacent to any of the salivary glands, but predominantly involve the parotid. A very small number are congenital. A few may be classified as cystic. Adenolymphomas are adjacent to large salivary glands and are slow-growing, encapsulated and indistinguishable from typical mixed tumors. The latter constitute the largest and most important group of salivary tumors and appear to be epithelial. They may or may not be malignant, and are classified only with difficulty. Malignant lesions form an ill-defined group, but approximately 40 per cent of reported cases have been placed in this class. They are characterized by rapid progress, pain, interference with motion of the jaw, destruction of adjacent tissues and metastasis. Treatment by surgical measures and irradiation should be individualized. Recurrence following the usual operations is found in from 20 to 45 per cent.—*Brit. J. Radiol.*, 17:3, 1944.

H. B. McCauley.

Rheumatic Fever: Diet as a Predisposing Factor

D. C. Peete

There is evidence that poor diet and lack of sunshine are important factors in rheumatic fever. The incidence is higher among people who live in cold, damp climates and who receive little sunlight. It is especially increased by these deficiencies when they are not compensated by diets abundant in protein, fish oils and dairy products. A study of seventy-six patients with rheumatic heart disease and acute rheumatic fever revealed that the average family size was 7.5 persons and suggested that such low-income group families have inadequate diets. There have been few recurrences of infection in patients who have improved their diets and have added to it high potency cod liver oil.—*Ann. Int. Med.*, 21: 44, July 1944.

Paul H. Keyes.

Studies on the Photodynamic Action of the Buccal Mucosa. I. Relationship Between the Photodynamic Action of the Buccal Mucosa and the Reticulo-Endothelial System

C. Jo.

The buccal mucosa of rabbits was irradiated with the visible spectrum of sunlight or carbon arc light after the intravenous injection of 0.5 per cent solution of trypaflavine. Local hyperemia, swelling or eruption always appeared, its severity depending on the dosage of the drug and the length of exposure to light. The reaction increased in degree when the reticulo-endothelial system was stimulated by an intravenous injection of India ink and decreased when the reticulo-endothelial system was obstructed.

The author believes that the normal blood stream contains a small amount of a substance sensitive to photodynamic stimulation, whose active principle seems to be generated in the reticulo-endothelial system scattered throughout the body, but not in the spleen since extirpation did not affect the reaction. The substance secreted by the glandular cells in the submucosa may contribute to the reaction.—*Chosen Igakkai Zassi (J. Korean M. A.)*, 29:2120-2132, 1939. (Abstr. *Far Eastern Sc. Bull.*, 4:3, March 1944.)

J. E. GILDA.

Cancer of the Lip

S. J. DOUGLAS

Of seventy-one consecutive cases of lip cancer treated at St. Anne's Hospital, Dublin, in 1939, seventy were in men, the ages ranging from 32 to 81 and averaging 62 years. The disease occurred predominantly in men engaged in farming or other outdoor occupations. Oral sepsis, irritating dental stumps, pipe smoking and exposure to wind and sun were important etiologic considerations. Differential diagnosis from tuberculous ulceration and syphilis was found essential. On admission, the average duration of symptoms was eight months, though in some it was five or six years. Thirty-eight per cent revealed submental or submaxillary lymph gland involvement. Early lesions were effectively treated with contact x-ray therapy. Surgery was most effective in adenopathic or metastatic cases. Three years of follow-up indicates complete cure in 53 per cent of the adenopathic cases.—*Brit. J. Radiol.* 17:185, 1944.

H. B. MCCAULEY.

Bone Degeneration in the Temporomandibular Joint

J. W. MCLAREN

The author presents the case of a man 39 years of age who had spent fifteen years as a sparring partner and had incurred a circular area of osseous degeneration beneath the articular surface of the mandible visible in roentgenograms of the temporomandibular joint. No other bone changes were observed. Operation for removal of the affected part disclosed a mucoid deposit in the superior aspect of the condyle. Pain over the joint and limitation of jaw movement had prompted the patient to seek medical attention. The operation completely eliminated the complaint.—*Brit. J. Radiol.*, 17:94, 1944.

H. B. MCCAULEY.

Adamantoblastomas in the Slye Stock of Mice

E. V. ZEGARELLI

The heads of seventy-nine mice having adamantoblastoma of the jaw were obtained from the Maud Slye colony at the University of Chicago. Each animal had been allowed to live its normal life span. Gross examination of the heads showed distortion, with malocclusion of the incisors resulting in most cases. Only the mandibular molar teeth appeared displaced by growing tumor masses. The tongue was often grossly misplaced and was frequently invaded by the neoplasms. Roentgenographic examination revealed monocystic involvement to be most frequent. The histopathology of the growths is amply described. Stratified squamous cells lined most of the cysts. The solid epithelial portions of the growths varied in quantity and in cell type.

It was concluded that (1) odontogenic cysts represent a stage in the development of the adamantoblastoma, (2) variations in type of pure tumors are not independent varieties, but must be considered as stages in the development of these growths, (3) enamel is never formed in pure adamantoblastomas, (4) the point of origin of these tumors in the jaw is the group of embryonal cells comprising the outer epithelial layer of the enamel organ and (5) adamantoblastomas are not characterized by the features usually ascribed to benign growths. Therefore, they should be designated as locally malignant with a slight tendency to metastasize.—*Am. J. Path.*, 20:23, January 1944.

S. J. FANALE.

Inheritance of Susceptibility and Resistance to Caries in Albino Rats (Mus Norvegicus)

H. R. HUNT AND F. A. HOPPERT

One hundred and nineteen rats (aged 35 days) from three local sources were placed on a caries-producing diet consisting of 66 per cent coarsely ground polished rice, 30 per cent whole milk powder, 3 per cent alfalfa meal and 1 per cent sodium chloride. Seventy per cent of the rice used was re-

tained on a 20-mesh screen. The lower molar teeth were examined every fourteen days. The size and location of carious lesions were recorded. The 116 surviving animals showed cavities at from 28 to 209 days after the experimental diets were begun. Susceptible rats from this first generation were crossed to start the susceptible strain, while resistant rats were paired to begin a resistant line. Selection, progeny testing and close imbreeding had been used since the second generation to build these two lines.

The mean of sibship averages (five or more rats each) for the first appearance of caries in the susceptible line steadily declined until it now fluctuates between twenty and thirty days. The eleventh generation consisted of seventy-four rats, the caries development requiring from fourteen to sixty-seven days with only six animals developing cavities later than forty days. The means of the resistant lines increased from 116 days in the second generation to 248 days in the sixth generation. Variability within this line is very high and as yet gives no indication of decreasing. The data suggest that a caries-immune strain may be attained.—*J. Am. Coll. Dentists,* 11:33, March 1944.

J. E. GILDA.

Absence of Correlation in New Zealand Between Blood Groups and Dental Decay, Allergy and Certain Character Traits

E. PHILIPP

Blood groups were related to medical grading, dental decay and type of personality in a group of 1640 New Zealand soldiers without finding any significant correlation. No attempt was made, however, to present the data upon which the findings were based. —*Am. J. Phys. Anthrop.,* 2 (New Series): 147-149, June 1944.

J. E. GILDA.

Ganglion Cell Tumors of the Neck

M. D. BOSSE, W. S. NETTROUR, J. A. SOFFEL AND J. Z. ROHM

Ganglion cell tumors in general and those of the neck in particular are infrequent. A generally accepted theory for the origin of these tumors is that they arise by multiplication of embryonal neurocytes, which continue to multiply and differentiate through the various stages up to adult ganglion cells. Two cases are reported. One patient was a white girl, aged 5½ years, with a well-differentiated tumor, a ganglioneuroma. She has remained well for three years since its removal. The other patient was a white man, aged 88, with a partially differentiated tumor, a ganglioneuroblastoma. This tumor was associated with roentgenologic evidence of pulminary metastases and a malignant clinical course.—*Am. J. Surg.,* (N.S.), 63: 120, July 1944.

J. E. GILDA.

Vincent's Infection: Observations and Conclusions Regarding the Etiology and Treatment of 1,017 Civilian Cases

A. F. STAMMERS

STUDY of a series of 1,071 civilian cases of Vincent's infection revealed an apparent seasonal variation in incidence and correlation with periods of intense enemy aerial activity and drives for industrial production. Persons from 19 to 30 years of age formed 67 per cent of total cases. Less than 4 per cent were under 14 or over 40. The sexes were equally affected. No history of contact or association with other affected persons could be obtained in 956 patients. Predisposing oral conditions were observed in 1,014 cases. Healthy mouths are apparently immune to the disease. Most cases are of autogenous origin. Fusospirochetal organisms were present in all cases. Treatment consisted of mechanical cleansing of the teeth and mouth. Aniline dyes, chromic acid, peroxide, and zinc oxide packs were also found useful. Vitamin therapy produced a response in only 17.8 per cent of cases.—*Brit. D. J.,* 76:147, 1944.

PAUL H. KEYES.

Anatomy of the Tongue of the Domestic Norway Rat

H. S. FISH, P. D. MALONE and C. P. RICHTER

RECENT developments in nutrition have revived interest in the anatomy and physiology of the tongue. Little or nothing is known regarding the anatomic basis of taste defects and the inheritance of morphologic characteristics of the tongue.—*Anat. Rec.,* 89:429, 1944.

PAUL H. KEYES.

ANNOUNCEMENTS

ANNOUNCEMENTS must be received by the fifth of the month in order to be published in the forthcoming issue of THE JOURNAL.

CALENDAR OF MEETINGS

ALPHA OMEGA FRATERNITY, New York, December 4-8.

AMERICAN ACADEMY OF RESTORATIVE DENTISTRY, Chicago, February 10-11, 1945.

DENTAL PROTECTIVE ASSOCIATION OF THE UNITED STATES, Chicago, December 18.

DETROIT DISTRICT DENTAL SOCIETY, Detroit, Mich., November 9.

DISTRICT OF COLUMBIA DENTAL SOCIETY, second Tuesday in each month, at the Medical Society Auditorium, Washington.

MID-CONTINENT DENTAL CONGRESS, St. Louis, Mo., October 30-November 1.

MID-CONTINENT DENTAL COUNCIL, St. Louis, Mo., October 30-November 1.

NATIONAL BOARD OF DENTAL EXAMINERS, December 4-5.

GREATER NEW YORK DENTAL MEETING, December 4-8.

NEW YORK SOCIETY OF ORTHODONTISTS, New York, November 13-14.

STATE SOCIETIES

November

Florida, at Miami Beach (9-11)
Ohio, at Columbus (12-15)

February 1945

Minnesota, at Minneapolis (27-March 1)

March 1945

Wisconsin (19-21)

April 1945

Kentucky, at Louisville (2-4)
Louisiana, at New Orleans (24-26)
Michigan, at Detroit (16-18)
Oklahoma, at Oklahoma City (15-18)
South Carolina, at Columbia (22-24)

May 1945

Connecticut, at Bridgeport (2-4)
Illinois, at Peoria (7-10)
Massachusetts, at Boston (14-17)
Mississippi, at Jackson (6-8)
Vermont, at Burlington (23-24)

June 1945

Washington, at Spokane (21-23)

STATE BOARDS OF DENTAL EXAMINERS

California, at San Francisco, November 6. Kenneth I. Nesbett, 515 Van Ness St., San Francisco 2, Secretary.

Colorado, at Denver, December 11-15. Ezra E. Schaefer, 724 Republic Bldg., Secretary.

Connecticut, at Hartford, November 14-18. C. G. Brooks, New London, Recorder.

Indiana, at Indianapolis, December 11-14. C. A. Frech, 504 Broadway, Gary, Secretary.

Texas, at Dallas, November 27-30. R. T. Weber, 311 Norwood Bldg., Austin 16, Secretary.

ALPHA OMEGA FRATERNITY

THE Alpha Omega Fraternity will have its headquarters at the Pennsylvania Hotel during the Greater New York Meeting, December 4-8. A luncheon will be given December 8 at 1 p.m.

SIDNEY SORRIN, *Chairman,*
745 Fifth Ave.,
New York 22, N. Y.

AMERICAN ACADEMY OF RESTORATIVE DENTISTRY

THE American Academy of Restorative Dentistry will hold its sixteenth annual meeting February 10-11, 1945, at the Stevens Hotel, Chicago.

W. SAMUEL TUCKER, *Secretary,*
940 Canal Bldg.,
New Orleans 12, La.

DENTAL PROTECTIVE ASSOCIATION OF THE UNITED STATES

THE annual meeting of the Dental Protective Association of the United States will be held at the Palmer House, Chicago, December 18 at 5 p.m.

ELMORE W. ELLIOT, *Secretary,*
104 Wesley Ave.,
Oak Park, Ill.

DETROIT DISTRICT DENTAL SOCIETY

THE Third Annual Dental Review of the Detroit District Dental Society will be held at the Hotel Statler, Detroit, November 9. Limited attendance clinics will be held at 10, 2 and 4 o'clock, with dinner at 6, followed by table clinics and movies.

R. W. WALMOTH, *Chairman,*
4421 Woodward Ave.,
Detroit 1, Mich.

DISTRICT OF COLUMBIA DENTAL SOCIETY

THE District of Columbia Dental Society holds it meetings on the second Tuesday of every month, at the Medical Society Auditorium, 1718 M. St., N.W., Washington.

J. WALTER BERNHARD, *Secretary,*
1835 Eye St., N.W.,
Washington.

MID-CONTINENT DENTAL CONGRESS

THE annual Mid-Continent Dental Congress, sponsored by the St. Louis Dental Society, will be held at the Hotel Jefferson, October 30-November 1.

HENRY F. WESTHOFF, *Chairman,*
Publicity Committee,
Missouri Theater Bldg.,
St. Louis, Mo.

MID-CONTINENT DENTAL COUNCIL

A SPECIAL invitation to attend the Mid-Continent Dental Council at St. Louis October 30-November 1 is extended to those dentists serving in the armed forces who are at this time stationed in the United States. Those planning to attend are requested to contact Capt. K. C. Marshall, O'Reilly General Hosptial, Springfield, Mo., so that suitable arrangements may be made.

RALPH B. RODE, *President.*

NATIONAL BOARD OF DENTAL EXAMINERS

THE National Board of Dental Examiners will hold its next sessions for the examination of candidates in Part 1 and 2 December 4-5 in schools where there are five or more candidates. Applications should come through the deans. For further information, address

GORDON L. TEALL, *Secretary,*
Box 71,
Hiawatha, Kan.

GREATER NEW YORK DENTAL MEETING

THE twentieth annual Greater New York Dental Meeting will be held December 4-8, at the Hotel Pennsylvania, New York City, Comprising the program are twenty-four morning registered clinics, twenty afternoon registered clinics, forty-five table clinics and thirty topic discussions; with thirty-two speakers, seven on medical-dental subjects, and twenty-six visual education films. The dental manufacturers, supply houses and laboratories will have exhibits of the newest materials and equipment available. Programs will be available in early November. Dentists in attendance should bring with them their A.D.A. membership cards to avoid confusion in registering.

NEW YORK SOCIETY OF ORTHODONTISTS

THE next meeting of the New York Society of Orthodontists will be held at the Waldorf-Astoria Hotel, New York, November 13-14.

NORMAN L. HILLYER, *Secretary,*
Professional Bldg.,
Hempstead.

CONNECTICUT STATE DENTAL ASSOCIATION

THE eighty-first annual meeting of the Connecticut State Dental Association will be held at the Hotel Stratford, Bridgeport, May 2-4, 1945.

EARLE S. ARNOLD, *Secretary.*

FLORIDA STATE DENTAL SOCIETY

THE Florida State Dental Society will meet November 9-11 at the Sea Isle Hotel, Miami Beach. The theme of the meeting will be maxillofacial surgery. Permission has been granted from Washington for travel involved in attending the meeting. The officers of the society are: president: E. C. Lunsford; president-elect: Fred O. Conrad; first vice-president: William P. Wood, Jr.; second vice-president: F. P. Meyer; secretary-treasurer: H. L. Cartee; librarian: Leland Daniels.

HORACE L. CARTEE, *Secretary,*
810 Huntington Bldg.,
Miami.

KENTUCKY STATE DENTAL ASSOCIATION

THE eighty-fifth annual meeting of the

Kentucky State Dental Association will be held at the Brown Hotel, Louisville, April 2-4, 1945.

J. L. WALKER, *Secretary,*
640 Barbee Way South,
Louisville.

ILLINOIS STATE DENTAL SOCIETY

THE next meeting of the Illinois State Dental Society will be held May 7-10 at the Pere Marquette Hotel, Peoria.

L. H. JACOB, *Secretary,*
634 Jefferson Bldg.,
Peoria 2.

LOUISIANA STATE DENTAL SOCIETY

THE Louisiana State Dental Society will hold its annual meeting in New Orleans April 24-26 at the Roosevelt Hotel.

F. HAROLD WIRTH,
General Chairman,
Local Arrangements Committee,
829 Maison Blanche Bldg.,
New Orleans 16.

MASSACHUSETTS DENTAL SOCIETY

THE 1945 meeting of the Massachusetts Dental Society will be held at the Statler Hotel, Boston, May 14-17.

P. E. ADAMS.

MICHIGAN STATE DENTAL SOCIETY

THE eighty-ninth annual meeting of the Michigan State Dental Society will be held April 16-18 at the Hotel Statler, Detroit.

S. G. APPLEGATE,
General Chairman,
630 E. Jefferson Ave.,
Detroit.

MINNESOTA STATE DENTAL ASSOCIATION

THE sixty-second annual session of the Minnesota State Dental Association will be held in the Municipal Auditorium, Minneapolis, February 27-March 1.

L. M. CRUTTENDEN, *Secretary,*
498 Lowry Medical Arts Bldg.,
St. Paul.

MISSISSIPPI DENTAL ASSOCIATION

THE Mississippi Dental Association will hold its next annual meeting in Jackson at the Robert E. Lee Hotel, May 6-8.

O. L. COLEE, *Secretary,*
Magnolia.

OHIO STATE DENTAL SOCIETY

THE seventy-ninth annual meeting of the Ohio State Dental Society will be held in the Neil House Hotel, Columbus, November 12-15. "Clinics for the general practitioner" have been arranged under four heads: table, limited attendance, question and answer discussions and exhibitors'. There will be the usual entertainment features. All A.D.A. members are invited.

EDWARD C. MILLS, *Secretary,*
255 E. Broad St.,
Columbus.

OKLAHOMA STATE DENTAL SOCIETY

THE Oklahoma State Dental Society will hold its 1945 meeting in Oklahoma City April 15-18.

E. W. Wise, *Secretary.*

COLLEGE OF SURGEON DENTISTS OF PUERTO RICO

THE College of Surgeon Dentists of Puerto Rico will hold its next meeting in December.

JOSE O. PORRATA, *Secretary.*

SOUTH CAROLINA STATE DENTAL ASSOCIATION

THE seventy-fifth, "Diamond Jubilee," annual meeting of the South Carolina State Dental Association will be held at the Columbia Hotel, Columbia, April 22-24, 1945.

C. M. DOUGLAS, *President,*
State Hospital,
Columbia.

VERMONT STATE DENTAL SOCIETY

THE next annual meeting of the Vermont State Dental Society will be held at the Van Ness Hotel, Burlington, May 23-24.

WASHINGTON STATE DENTAL ASSOCIATION

THE next meeting of the Washington State Dental Association will be held at Spokane, June 21-23.

G. D. WILLIAMS, *President,*
511 Paulsen Bldg.,
Spokane.

WISCONSIN STATE DENTAL SOCIETY

THE 1945 meeting of the Wisconsin State Dental Society, the Diamond Jubilee, will be held March 19-21, 1945.

BOARD OF DENTAL EXAMINERS OF CALIFORNIA

THE next examination to be conducted by

the Board of Dental Examiners of California for licensure in California will commence November 6 at the Physicians and Surgeons College of Dentistry, San Francisco. Applications should be filed with the office of the secretary at least twenty days before the examination. Address all correspondence to

KENNETH I. NESBETT, *Secretary,*
515 Van Ness Ave.,
San Francisco 2.

COLORADO STATE BOARD OF DENTAL EXAMINERS

THE Colorado State Board of Dental Examiners will hold an examination in Denver December 11-15. All credentials and fees must be filed with the secretary at least two weeks prior to the examination.

EZRA E. SCHAEFER, *Secretary,*
724 Republic Bldg.,
Denver.

CONNECTICUT DENTAL COMMISSION

THE Connecticut Dental Commission will meet in Hartford November 14-18 for the examination of applicants for license to practice dentistry or dental hygiene. Applications should be in the hands of the recorder at least ten days before the meeting. For application blanks and further information, apply to

C. G. BROOKS, *Recorder,*
New London.

INDIANA STATE BOARD OF DENTAL EXAMINERS

THE next meeting of the Indiana State Board of Dental Examiners will be held in Indianapolis, December 11-14. All applications must be in the hands of the secretary at least two weeks prior to the date of examination. For applications or information, address

C. A. FRECH, *Secretary,*
504 Broadway,
Gary.

TEXAS STATE BOARD OF DENTAL EXAMINERS

THE Texas State Board of Dental Examiners will hold an examination in Dallas November 27-30, with headquarters at the Baker Hotel. Because this period includes the Thanksgiving holiday, the usual order will be reversed, the practical part of the examination being given the first two days and the written the last two. The deadline for accepting applications is October 27. Application blanks and additional information will be furnished on application to the Executive Office, 311 Norwood Bldg., Austin 16.

R. T. WEBER, *Secretary.*

ALPHA OMEGA

ALL Massachusetts members of Alpha Omega in service, both in this country and abroad, are requested to communicate with Abraham Gurvitz, War Service Committee, Alpha Omega Fraternity, 371 Commonwealth Ave., Boston 15, Mass.

SAMUEL S. HIRSON, *Secretary,*
Boston Alumni Chapter,
471 Commonwealth Ave.,
Boston, Mass.

DENTAL INTERNSHIPS AND FELLOWSHIPS

THE Walter G. Zoller Memorial Dental Clinic, University of Chicago, offers rotating internship appointments. The clinic maintains four divisions of service: 1. The Zoller Clinic in Billings Hospital for all types of dental service. 2. The University Dental Clinic in Billings Hospital for diagnosis and minor oral surgery. 3. The Prenatal Dental Clinic in Chicago Lying-In Hospital for general service. 4. The Orthopedic Dental Clinic in the Home for Destitute Crippled Children for general dentistry. In addition to clinical practice, the intern is assigned to the department of anesthesia for instruction in the administration of general anesthetics. Opportunity is afforded to witness major surgical procedures about the face and jaws.

The Zoller clinic offers fellowships to those who desire advanced training in the basic sciences and who wish to equip themselves for dental teaching and research. Appointments carry liberal stipends and free tuition.

Interns and fellows have the privilege of attending all departmental seminars. Those interested should write for application blanks to the Director, Walter G. Zoller Memorial Dental Clinic, University of Chicago, Chicago 37, Ill.

INTERNSHIP AND VOLUNTEER EXTERNSHIPS

THERE is an internship, as well as several volunteer externships, available for recent graduates in the Dental Department of the Bronx Hospital. Opportunity is offered for training and experience in oral surgery and exodontia, orthodontia, periodontia, endodontia (root-canal therapy) and operative dentistry. Application forms can be obtained by communicating with S. Berton Gerstner, Director, Dental Department, Bronx Hospital, Fulton Ave. at 169th St., Bronx 56, N. Y.

INTERNSHIPS AND RESIDENCY

Two dental intern and one dental resident vacancies are announced by the St. Louis City Hospital, appointments available immediately. Address Leo J. Wade, M.D., medical director, Office of City Hospital, 1515 Lafayette Ave., St. Louis, Mo.

DENTAL INTERNSHIPS

THE Greater New York Conference of Hospital Dental Services, comprised of a group of men who are chiefs of dental services, would like to aid those men seeking dental internships in hospitals in the metropolitan area. Those who are not in the Army or the Navy and who are desirous of obtaining internships should communicate with

MARY E. REILLY, *Secretary*,
386 Fourth Ave.,
New York, N. Y.

DENTAL INTERNSHIPS

AN internship is available to a recent dental graduate at the Research and Educational Hospital of the University of Illinois Medical Center. An unusual opportunity is afforded for training and experience in hospital dentistry and the medical aspects of dental diseases. Application forms can be obtained from the Dean, University of Illinois College of Dentistry, 808 South Wood St., Chicago 12, Ill.

CARNEGIE FELLOWSHIPS FOR RESEARCH AND GRADUATE WORK IN THE DENTAL SCIENCES

AIDED by the Carnegie Corporation of the New York and the Eastman Dental Clinic, the School of Medicine and Dentistry of the University of Rochester offers scholarships paying $1,200-$1,800 per annum to well-trained dental graduates who are interested in research or who desire special training in the basic medical sciences as an aid in dental teaching or practice. A few fellowships also offer part-time training in oral surgery. On appointment, the applicant is given the title of Fellow in Dentistry and, according to his interests, works as a graduate student in one of the departments of the medical schools. Here, excellent laboratory facilities, adequate direction and close contact with various investigations related to medicine and dentistry help his research and study. Fellows are encouraged to attend medical classes, seminars and staff meetings and, when opportunity presents itself, to participate in class instruction and scientific meetings. Contact with the problems of clinical dentistry may be maintained through the Oral Surgery Clinic of the Strong Memorial Hospital and the Rochester Dental Dispensary. With the approval of the Graduate Council of the university, Fellows may prepare themselves for the degree of M.S. or Ph.D. Applications should be made to

GEORGE H. WHIPPLE, *Dean*,
Rochester, N. Y.

FEDERAL POSITIONS FOR DENTAL HYGIENISTS

THE United States Civil Service Commission is continuing to recruit dental hygienists for federal positions. Experience is no longer necessary.

Completion of a full course leading to graduation from a recognized school of oral hygiene and registration as a dental or oral hygienist in a state, territory or the District of Columbia will qualify applicants.

Students enrolled in schools of oral hygiene may apply for federal positions when they are within nine months of completion of the course and may enter on duty on completion and registration. There are no age limits for the positions and no written tests. Appointees receive $1,970 a year entrance salary, which incorporates compensation for eight hours of overtime included in the present forty-eight hour federal work week. The majority of positions are in Army hospitals and dispensaries throughout the country, particularly in the West and Southwest. Vacancies may occur also in the Veterans Administration and the Public Health Serv-

ice. Applications should be filed with the Civil Service Commission, Washington 25, D. C.

W. C. HULL, *Executive Assistant,*
United States Civil Service Commission,
Washington, D. C.

CHICAGO DENTAL SOCIETY FOURTH ANNUAL PRIZE ESSAY COMPETITION

To encourage continued scientific research in all phases of dentistry, the Chicago Dental Society again offers a cash prize of $500 to the author of the most meritorious essay reporting an original investigation and containing new and significant material of value to dentistry.

The winner of the award will be invited to present his essay at the eighty-first Midwinter Meeting of the Chicago Dental Society, to be held in Chicago during February 1945. The expenses of the winner will be paid by the society and will be in addition to the prize of $500.

Any member of the American Dental Association or of a recognized foreign dental society, or any one affiliated with a recognized institution in the dental field may apply for authorization to enter the competition. The contest is not limited to research men having technical staffs and elaborate equipment at their disposal, but is open to any qualified person who believes that he has something new and important to contribute to dentistry. The rules are simple and fair and all contestants, whether students, teachers or practitioners, have an equal opportunity.

Application forms and contest rules can be obtained by writing to the Chicago Dental Society, 30 N. Michigan Ave., Chicago 2, Ill. All applications must be filed in the office of the society by October 2, 1944.

PRIZE ESSAY CONTEST, AMERICAN ASSOCIATION OF ORTHODONTISTS

THE Research Committee of the American Association of Orthodontists is empowered by the board of directors to conduct a prize essay contest. The prize has been set at $200 and will be offered annually until further notice. The terms of the competition are as follows:

Eligibility: Any student enrolled in a recognized university, or any person who has completed his or her formal education in orthodontia not more than two years prior to January 1, 1945, is eligible to compete for the prize.

Essay: The essay must represent a piece of original research having a direct bearing on the field of orthodontia. It may relate either to a biologic or a clinical problem and may represent material that has been offered in partial fulfilment of the requirements of a graduate or postgraduate degree, or any graduate, postgraduate or undergraduate contest. No papers previously submitted for publication or in press will be accepted. All essays must be in the hands of the committee not later than two months prior to the annual meeting of the association. Consult the periodic literature for this date. If no essay is deemed worthy by the committee, the prize will be withheld.

Award: The prize-winning essay will be accorded a place on the scientific program of the annual meeting of the association, at which time the prize will be awarded. The association will retain publication rights of the first three choices. For further information, address

ALLAN G. BRODIE, *Chairman,*
Research Committee, A.A.O.
30 North Michigan Ave.,
Chicago 2, Ill.

COURSE OF INSTRUCTION IN INDUSTRIAL HYGIENE

COLUMBIA UNIVERSITY, through the DeLamar Institute of Public Health, announces intensive instruction in the dental aspects of industrial hygiene during the week beginning December 11. The curriculum is intended for those responsible for or interested in the dental aspects of industrial hygiene, for dentists already working in this field and for those who foresee the probability of being associated with future developments in this phase of dental practice. Instruction therefore is designed to introduce the dentist to the general field of industrial hygiene and to give him an appreciation of the entire problem of industrial health so that he may better integrate the dental aspects of this subject in the general industrial health program. Members of the DeLamar Institute of Public Health, the School of Dental and Oral Surgery and others from the College of Physicians and Surgeons, Columbia University, with guest lecturers, will constitute

the faculty. Attendance will be limited to twenty-five. Persons other than dentists, in exceptional circumstances, may be admitted. Applications will be considered in the order received. The registration fee will be $25. For further information, address the Director, DeLamar Institute of Public Health, 600 W. 168th St., New York 32, N. Y.

GRADUATE SCHOOL OF MEDICINE, UNIVERSITY OF PENNSYLVANIA

THE Graduate School of Medicine of the University of Pennsylvania, organized in 1916 to provide for graduate studies in clinical medicine, has enlarged the scope of its activities to include graduate studies in dentistry, according to announcement by George William McClelland, president of the university. The graduate dental studies will be under the direction of John W. Ross, who has been appointed vice-dean for dentistry in the graduate school of medicine. He will work in cooperation with the vice deans for other medical studies under Robin C. Buerki, dean of the graduate school of medicine. In connection with the new graduate program in dentistry, courses are being planned for graduate studies in oral surgery, orthodontics, prosthetics and oral medicine-periodontics, according to Dr. Buerki.

The first period of study for those students entering on the graduate program will be devoted to basic studies involving the medical sciences as applied to the clinical specialties concerned, together with the principles and practice of that specialty, and its relation to other clinical specialties. The studies of this period will lead to a certificate.

The second period of study will be under preceptors and this period must be preceded by the basic studies at the university. The studies under the preceptors may be carried on at any approved institution and academic credit will be granted for them by the university, provided the specific plan for such studies for each candidate receives due approval and acceptance by the university. The successful completion of the period of study carried on under the preceptors will lead to the degree of master of science in dentistry.

Dr. Buerki pointed out that the program in dentistry in the graduate school of medicine will be independent of the school of dentistry at the university, but that the two schools will work in complete accord, and facilities and faculty of the school of dentistry will be available for the graduate studies and special work in the respective clinical fields.

Dr. Ross, who has long been prominent in the dental profession, was graduated from the school of dentistry at the University of Pennsylvania in 1917 and was an instructor in orthodontics there from 1918 to 1924. He taught the advanced course in orthodontics at Columbia University from 1929 to 1939.

UNITED STATES PATENTS APPLICABLE OR PERTAINING TO DENTISTRY ISSUED DURING AUGUST AND SEPTEMBER 1944

August 1

2,354,649, ROBERT E. BRUCKNER. Dental syringe.

2,354,780, HOWARD T. STEWART. Dental appliance.

2,355,012, GUY C. REIFSNYDER. Mouth spray syringe.

August 8

2,355,231, CLIFTON E. MOORE. Apparatus and process for sterilizing and destroying epithelial tissues.

August 22

2,356,447, ALLEN BERNARD CLINE. Denture repairing device.

2,356,513, PAUL JEAN JACQUES GONON. Process for increasing the retention capacity of the sealing surface of artificial porcelain teeth.

September 5

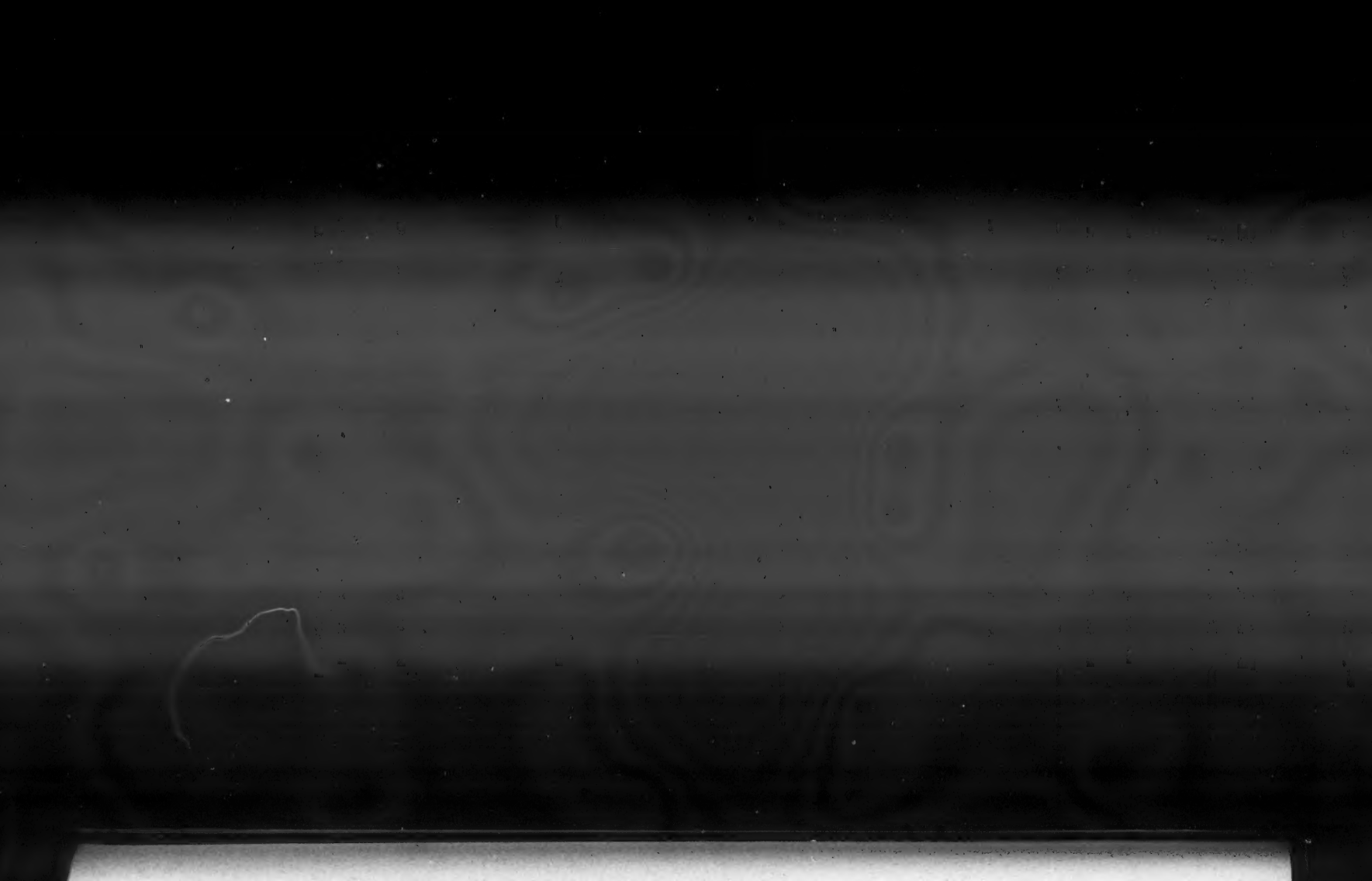
2,357,292, ARTHUR W. STAHL. Sterilizer for hypodermic needles.

2,357,757, SIMON MYERSON. Method of making artificial teeth.

September 12

2,357,969, FRANK T. POWERS. X-ray cassette.

September 19

2,358,645, WILLIAM J. KIFF. Dental device.

2,358,730, JOSEPH F. NELSON & HENRY G. NAUSS. Method for forming artificial dentures.